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A TEXT BOOK

MEDICAL JURISPRUDENCE

FOR

INDIA

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BY

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PREFACE.

In compiling this book my object has been to supply a want which I have often felt while lecturing on Medical Jurisprudence in an Indian college, viz. that of a concise manual of the subject specially adapted for use in India. In doing so I have made the freest use of the numerous standard treatises on Medical Jurisprudence and subjects allied to it, and of the admirable Bengal Medico-legal Reports of my friends Surgeons-Major K. McLeod and R. Harvey. So freely have I drawn from such works, that it is possible I may have in places omitted to acknowledge my indebtedness to them; if so, I trust that my omission will be considered repaired by this general acknowledgment.

I have also to thankfully acknowledge the personal assistance freely accorded to me by many of my friends, and especially the very great and valuable aid given me by Mr. J. D. Inverarity, barrister-at-law, Brigade Surgeon W. Dymock, and Surgeon-Major K. R. Kirtikar. I have further to express my thanks to Colonel Boyd, Bo. S. C., from whose sketches, and Dr. Heneage Gibbes, from whose micro-photographs, the illustrations of this book are derived. Any success this work may have I am certain will be largely due to the help my friends have so freely accorded to me.

I. B. LYON.

Grant Medical College, Bombay
September 1888.

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BENGAL SECRETARIAT

MEDICAL JURISPRUDENCE.

CHAPTER I.

INTRODUCTORY. OFFENCES AND THEIR PUNISHMENT. CONSTITUTION AND PROCEDURE OF CRIMINAL COURTS. LAW OF EVIDENCE.

THE term Medical Jurisprudence, as now commonly used, may be taken as synonymous with Forensic Medicine, or "that branch, of medical science which treats of things medical in relation to evidence and judicial procedure." A medical jurist may have to deal with

A. Civil Cases, *e.g*, Marriage, Legitimacy, Inheritance, &c., or—

B. Criminal Cases, *e.g.*, Murders, Assaults, Rape, &c.

In many of these cases, questions arise which have little to do with the ordinary work of a medical man in the detection and treatment of disease; *e.g* the detection of blood-stains, the character of wounds inflicted during life, minutiæ regarding poisons, &c. Hence the necessity of a special course of study of these and other matters likely to form the subject of inquiry in the course of a civil or criminal proceeding.

It is desirable that a medical jurist should possess a certain amount of knowledge of the law of evidence and judicial procedure, especially of the law relating to criminal cases, seeing that these form the great majority of the cases with which he has to deal. Hence a short sketch is desirable of the principal points relating to—I. Offences and their Punishment. II. The Constitution and Procedure of the Courts before which Offences may be inquired into, and III. The Law of Evidence.

I. OFFENCES AND THEIR PUNISHMENT.

In India, offences are defined, and the punishment awardable for each offence limited, by the **Indian Penal Code** (*Act* 45 *of* 1860), certain general provisions of which may be here considered.

A. Acts are not offences if they come under certain

general exceptions laid down in the Code. The principal of these are, that acts are not offences if done—

I. By a child under the age of seven. (Sect. 82.)

II. By a child between the ages of seven and twelve (not as in England between the ages of seven and fourteen), "who has not attained sufficient maturity of understanding to judge of the nature and consequences of his conduct on that occasion." (Sect. 83.)

III. By a person of unsound mind, "if by reason of unsoundness of mind the doer of the act is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law." (Sect. 84.)[1]

IV. By an intoxicated person, but only, provided,

1. "the thing which intoxicated him was administered to him without his knowledge or against his will;" and

2. when by reason of the intoxication so induced, the intoxicated person is "incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law." (Sect. 85.)

V. In good faith, and for the benefit of the person on whose body the act is done (under this exception come surgical operations), provided certain conditions are complied with, the chief of which are—

1. That the act must not be intended to cause death. (Sect. 88.)

2. That the act must not of itself be an offence independently of any harm it may cause to the person on whose body the act is done; *e.g.* causing miscarriage, except for the purpose of saving the life of the mother. (Sect. 91.)[2]

3. That the act is done with the **consent of the sufferer**, such consent,

a. Not being known to the doer of the act to have been given under fear of injury or misconception of fact.

b. Not having been given by a person under twelve years of age.

c. Not having been given by a person who by reason of unsoundness of mind or intoxication is unable to understand the nature and consequence of that to which he gives his consent. (Sect. 90.)

Consent of the sufferer may, however, be dispensed with, if—

a'. He is of unsound mind or under the age of twelve, if the consent of the person having lawful charge of him has been obtained (sect. 89); or

b'. The circumstances are such that it is impossible for him to signify his consent, and he has no person in lawful charge of him from whom it is possible to obtain consent in time for the thing to be done with benefit. (Sect. 92.)

[1] See also Insanity, p. 411. [2] See also Causing Miscarriage, p. 373.

But in those cases where consent is dispensed with, an act which is known to be likely to cause death or grievous hurt,[1] may only be done for the purpose of preventing death or grievous hurt, or for the curing of any grievous disease or infirmity. (Sect. 89.)

B. An offence may be committed by illegal omission (sect. 32); thus, a woman may commit murder by intentionally omitting to supply her infant with food.[2]

C. Attempts to commit offences are, in some cases, punishable under special sections of the Code, *e.g.*, an attempt to commit murder (sect. 307), or culpable homicide not amounting to murder (sect. 308), or suicide (sect. 309). Attempts not punishable under special sections of the Code, are dealt with by sect. 511: "Whoever attempts to commit an offence punishable by this Code with transportation or imprisonment, or to cause such an offence to be committed, and in such attempt does any act towards the commission of the offence, shall, where no express provision is made by this Code for the punishment of such attempt, be punished with transportation or imprisonment of any description provided for the offence, for a term of transportation or imprisonment which may extend to one-half of the longest term provided for that offence, or with such fine as is provided for the offence, or with both."[3]

D. The punishments awardable for offences are defined by sect. 53 of the Penal Code to be: 1. Death. 2. Transportation. 3. Penal servitude. 4. Imprisonment, which may be either (*a*) Rigorous, that is, with hard labour; or (*b*) Simple, that is, without hard labour. 5. Forfeiture of property. 6. Fine. And under Act VI. of 1864, whipping may be awarded for certain offences. The chief medico-legal points in connection with these punishments are—

I. **Death.**—This (see sect. 368 of Act X. of 1882, the Code of Criminal Procedure) must be by hanging. Pregnancy may be pleaded in bar of execution: "If a woman sentenced to death be found to be pregnant, the High Court shall order the execution of the sentence to be postponed, and may commute the sentence to transportation for life." (C. P. C., sect. 382.) In India the question as to by whom the existence or otherwise of pregnancy is to be determined appears to be left to the discretion of the Court. In England, according to an old rule of law, in such cases a jury of twelve matrons is empanelled and sworn, to try whether the "prisoner be with child, of a quick child."[4]

II. **Hard Labour.**—A medical man may be called on to determine whether a prisoner is in a fit state of health or not to perform certain descriptions of labour; in such a case, the chief points for inquiry would be as to—

[1] See Wounds, p. 56.
[2] See Infanticide, p. 368.
[3] See also Causing Miscarriage, p. 374.
[4] See Pregnancy, p. 332.

(1) The prisoner's general health.

(2) His freedom or otherwise from cardiac disease, aneurism, or grave disease of the respiratory organs.

(3) The nature of the labour (if any) he has previously been engaged on, and whether he has been gaining or losing weight.

(4) The proportion borne by the labour it is proposed to exact, to the weight of the individual, 2 foot-tons per 1 lb. of body-weight being an ordinary, and 3 foot-tons per 1 lb. of body-weight a very hard day's work. In many cases labour to be performed may be reduced to foot-tons by Haughton's formula, which may be stated as follows. Add together the body-weight of the individual (in pounds), and the weight (in pounds) carried by him; multiply this by the height (in feet) ascended, plus one-twentieth of the horizontal distance (in feet – 1 mile = 5280 feet) travelled, and divide the product by 2240.[1]

III. **Whipping.**—Here the principal points are—

(1) All females, and all males over forty-five years of age, are exempted. (C. P. C., sect. 393.)

(2) A medical man may be called upon to certify whether or no an offender is in a fit state of health to undergo this punishment. (C. P. C., sect. 394.)

(3) A medical man may, during the execution of a sentence of whipping, be called upon to certify as to the fitness, or otherwise, of the offender to undergo the remainder of the sentence, and should he certify that the offender is not in a fit state of health to undergo the remainder of the sentence, the whipping must be finally stopped, *i.e.*, the remainder of the sentence cannot at some future period be inflicted. (C. P. C., sect. 394.)

II. CONSTITUTION AND PROCEDURE OF CRIMINAL COURTS.

This is dealt with by the **Code of Criminal Procedure** (*Act X. of* 1882).

Section 6 of this Code prescribes that, besides the **High Courts**, there shall be five classes of Criminal Courts, viz.:—

1. **Courts of Session.**
2. **Courts of Presidency Magistrates.**
3. **Courts of Magistrates of the First Class.**
4. **Courts of Magistrates of the Second Class.**
5. **Courts of Magistrates of the Third Class.**

The powers of these Courts are defined in Chapter III. of the Code. Thus, of the various classes of courts, the High Courts and Courts of Session are empowered to try any offence, and to pass (except in the case of a Court of Session presided over by an Assistant Sessions Judge) any sentence authorized by

[1] See also Starvation, p. 107. 2240 lbs. = 1 ton.

law; but a sentence of death passed by a Court of Session shall be subject to confirmation by the High Court. (Sect. 31.) Next in grade come the Courts of Presidency Magistrates, and Courts of Magistrates of the first class. These Courts may not try certain grave offences, *e.g.*, murder, causing miscarriage, rape, and unnatural offences, and may not, for any single offence, sentence to more than two years' imprisonment and 1000 rupees fine. Courts of Magistrates of the second class may not try any offence punishable with three years' imprisonment (sect. 29), and may not, for any single offence, sentence to more than six months' imprisonment and 200 rupees fine. Courts of Magistrates of the third class may not try an offence punishable with one year's imprisonment, and may not, for any single offence, sentence to imprisonment for more than one month and 50 rupees fine. (Sect. 32.) Third-class Magistrates may not sentence to solitary confinement or to whipping, and Second-class Magistrates may only sentence to whipping, if specially empowered by the Local Government. (Sect. 32.)

Magistrates in addition to the powers they possess in respect to the trying and sentencing of offenders, also possess certain other powers (see sect. 36 and schedule III.), and may have various powers conferred upon them. Thus Presidency Magistrates and First-class Magistrates have power to commit an offender for trial before the High Court, or a Court of Session and other Magistrates also may be so empowered. Magistrates may also be empowered to hold inquests under sect. 176 of the Code.

Every criminal trial before a High Court must be by jury. (Sect. 267.) Juries are of two kinds—Special Juries and Common Juries. A special jury is composed of persons taken from a special list of two hundred. In every case where the offence to be tried is punishable with death, and also in such other cases as a Judge of the High Court may direct, the trial is to be before a special jury. (Sect. 276.) In all other cases, the trial takes place before a common jury, *i.e.*, composed of persons whose names appear in the general list of persons liable to serve as jurors. **Trials before a Court of Session** are ordinarily conducted with the aid of assessors, two or more in number, but the Local Government can, by order in the official *Gazette*, direct trials before any Court of Session of all offences or any particular class of offences to be by jury. Sect. 320 of the Code enumerates the persons exempt from liability to serve as jurors or assessors, and clause (*h*) of this section includes among those exempted "surgeons and others who openly and constantly practise the medical profession."

Sects. 464 and 465 provide that when an accused person appears to be "*of unsound mind, and consequently incapable of making his defence,*" the Magistrate holding the inquiry or trial, or the Court of Session or High Court, in the case of persons

committed for trial before these Courts, shall first inquire into or try the fact of such unsoundness of mind, and if this be proved, the inquiry or trial shall be postponed. Sect. 464 further directs that Magistrates in such cases shall cause the accused to be examined by the civil surgeon, or such other medical officer as the Local Government directs, and thereupon shall examine such surgeon or other officer as a witness, and shall reduce the examination to writing.

Inquiries into cases of unnatural or sudden death may be either—

(1.) **An inquiry held by an officer in charge of a police-station**, or in the Presidencies of Madras and Bombay by the head of the village, under sect. 174 of the Code; or,

(2.) **An inquest held by a Magistrate** under sect. 176 of the Code; or,

(3.) **An inquest held by a coroner** under the Coroners Act (Act IV. of 1871).

As regards (1), sect. 174 of the Code directs that every **officer in charge of a police-station** shall (or, in the Presidencies of Madras and Bombay, the head of the village may) on receiving information that a person has committed suicide, or been killed by another, or by an animal, or by machinery, or by an accident, or has died under circumstances raising a reasonable suspicion that some person has committed an offence, proceed to the place where the body of such deceased person is, and there, in the presence of two or more respectable inhabitants of the neighbourhood, shall make an investigation, and draw up a report of the apparent cause of death. The officer holding the inquiry is also authorized to forward the body, for examination, to the nearest medical officer appointed by the Local Government to conduct such examinations.

As regards (2), sect. 176 of the Code directs that any **Magistrate empowered to hold inquests** may hold one either instead of, or in addition to the inquiry conducted by a police officer or head of a village under sect. 174, and further, that the nearest Magistrate empowered to hold inquests, shall hold one in any case where a person dies while in custody of the police. Sect. 176 also gives power to such Magistrate to order exhumation of a body for examination.

As regards (3), in each presidency town there is an officer called the **coroner, whose duties** are thus defined in sect. 8 of Act IV. of 1871 (the Coroners Act): Where a coroner has reason to believe that the death of any person has been caused by accident, homicide or suicide, or suddenly by means unknown, or that any person being a prisoner has died in prison, and that the body is lying within the place for which the coroner has been appointed, the coroner shall inquire into the cause of death. The principal other provisions of the Coroners Act are :—

(*a'*) That an inquest need not be held in a case where a prisoner has died in prison from cholera or epidemic disease. (Sect. 9.)

(*b'*) That the coroner may order a body to be exhumed. (Sect. 11.)

(*c'*) That the inquiry is to be conducted with the aid of a jury, who, with the coroner, are to view the body. (Sects. 12 and 15.)

(*d'*) The coroner is empowered to order a post-mortem examination, with or without analysis of the viscera, to be made by any medical witness summoned to attend the inquest, and such medical witness, other than the chemical analyser to Government, shall be entitled to such reasonable remuneration as the coroner thinks fit. (Sect. 118.)

(*e'*) Evidence is to be given on oath (sect. 19), reduced to writing by the coroner (sect. 20), and the jury are to return a verdict (sect. 23).

(*f'*) A coroner may appoint a deputy coroner to act for him when sick, or absent for any lawful or reasonable cause. (Sect. 28.)

EVIDENCE.

In the words of Sir James Stephen, "Every judicial proceeding has for its purpose the ascertaining of some right or liability," and "all rights and liabilities are dependent on and arise out of facts." This being the case, it is for many reasons expedient that definite rules should be laid down whereby may be determined various points connected with the proof of such facts, such as: (1) What sort of facts are to be proved? (2) What sort of proof is to be given of these facts? (3) How such proof is to be given, &c., &c. Such rules constitute the law of evidence, and those applicable in India are embodied in the Indian Evidence Act (Act I. of 1872).

Some of the more important provisions of the Law of Evidence are as follows:—

I. The facts concerning which evidence may be admitted in the course of any judicial proceeding must be either (*a*) facts in issue or (*b*) relevant facts. (Sect. 5.)

Facts in issue may in general terms be defined to be the facts on which the right or liability in question directly depends: *e.g.*, at the trial of A for the murder of B, by beating him with a club with the intention of causing his death, the following are facts in issue:—

A's beating B with the club.

A's causing B's death by such beating.

A's intention to cause B's death. (Sect. 5.)

Relevant facts may in general terms be defined to be, facts which, though not themselves in issue, affect the probability of

facts in issue: *e.g.*, A is tried for the murder of B by poison, the following, although not facts in issue, are relevant facts:—

That A, immediately after B had swallowed the liquid alleged to have contained the poison, threw away the remainder of the liquid and washed the vessel which had held it.

That B was in good health previous to the occurrence of the symptoms attributed to poison.

That A before the death of B procured poison similar to that which was administered to B.

That B died from heart disease.

Chapter II. of the Indian Evidence Act defines the various modes in which a fact must be connected either with a fact in issue, or a relevant fact, in order to be of itself a relevant fact. Two of the sections of this chapter (45 and 46) are of special medico-legal importance.

These two sections refer to the opinions of skilled witnesses, or as they are technically called, **experts.** Sect. 45 defines an "expert" to be a person specially skilled in foreign law, science, art, or the identity of handwriting, and declares that in cases where the Court has to form an opinion upon a point of foreign law, science, art, or identity of handwriting, the **opinions of experts** are relevant facts. Sect. 46 states further that "Facts, not otherwise relevant, are relevant if they support or are inconsistent with the opinions of experts, when such opinions are relevant."

II. Some facts require no proof, *e.g.*, facts noticed by the Courts *ex officio*, and facts deemed notorious. Such facts are technically called, facts of which the Court "will take judicial notice." (Sect. 56.) Sect. 57 enumerates the facts of which a Court shall take judicial notice, *e.g.*, the divisions of time, the rule of the road on land or at sea, &c., &c., and empowers the Court on all such matters, and also on all matters of public history, literature, science, or art, to resort for its aid to appropriate books or documents of reference.

III. When facts are not such as the Court will take judicial notice of, or are not admitted by the parties to the proceeding before the Court, any relevant facts upon which either party desires the Court to act must be proved by evidence. Evidence is either oral or documentary, and by a proviso attached to sect. 60, if oral evidence refers to the existence or condition of any material thing, the Court may require the production of such thing for its inspection—*e.g.*, a blood-stained weapon or article of clothing, a portion of eliminated poison, &c., &c. Hence such matters should always after examination be, if possible, preserved for production before the Court. Sect. 293 of the Criminal Procedure Code and sect. 392 of the Civil Procedure Code also provide for the inspection by the jury or assessors in criminal cases, and by the Court in

civil cases, of certain localities, the inspection of which may assist in determining the question in dispute.

Sect. 60 directs that "**oral evidence must in all cases whatever be direct,**" that is to say—

If it refers to a fact which could be seen, it must be the evidence of a witness who says he saw it.

If it refers to a fact which could be heard, it must be the evidence of a witness who says he heard it.

If it refers to a fact which could be perceived by any other sense, or in any other manner, it must be the evidence of a witness who says he perceived it by that sense, or in that manner.

If it refers to an opinion, or to the grounds upon which that opinion is held, it must be the evidence of the person who holds that opinion on those grounds.

Certain exceptions, however, are permitted to the rule, that oral evidence must in all cases whatever be direct, the principal of which are as follows:—

(1) **The Opinions of Experts** expressed in any treatise commonly offered for sale, and the grounds upon which such opinions are held, may be proved by the production of such treatises, if the author is dead, or cannot be found, or has become incapable of giving evidence, or cannot be called as a witness without an amount of delay or expense which the Court regards as unreasonable. (Sect. 60.)

(2) **Dying declarations.** Statements, written or verbal, made by a person who is dead as to the cause of his death, or as to any of the circumstances of the transaction which resulted in his death, are admissible in cases where the cause of that person's death comes into question, no matter whether the person making such statement was or was not under expectation of death at the time of making it. (Sect. 32.) It may here be noted that, according to the law of England, a dying declaration is only admissible when the person making it is in actual danger of death, and is aware of his danger.

(3) **Evidence given in a previous judicial proceeding** by a witness who is dead, or cannot be found, or is incapable of giving evidence, or is kept out of the way by the adverse party, or whose attendance cannot be obtained without an amount of delay or expense which the Court under the circumstances of the case considers unreasonable, is admissible under certain conditions (see sect. 33). Under this rule the deposition on oath of a dying person taken by a magistrate in presence of the accused, becomes admissible in place of a dying declaration, and when the circumstances of the case permit, it is always advisable to take steps to obtain such a deposition.

(4) **The deposition of a medical witness,** taken and attested by a magistrate in the presence of the accused, may be given in evidence in any inquiry or trial or other proceeding under the

Code of Criminal Procedure, although the deponent is not called as a witness, but the Court may, if it thinks fit, summon and examine the deponent. (C. P. C., sect. 509.)

(5) **Any document purporting to be a report under the hand of the Chemical Examiner,** or assistant chemical examiner to Government, upon any matter or thing duly submitted to him for examination, or analysis and report, in the course of any proceeding under the Code of Criminal Procedure, may be used as evidence in any inquiry, trial, or other proceeding under the said code. (C. P. C., sect. 510.)

IV. Witnesses.—**All persons are competent** to testify unless the Court considers that they are prevented from understanding the questions put to them, or from giving rational answers to those questions, by tender years, extreme old age, disease, whether of body or mind, or any other cause of the same kind. (Sect. 118.)

In certain special cases (see sects. 121 to 129) a witness may on certain grounds **claim exemption** from being compelled to answer certain questions, *e.g.*, "no public officer shall be compelled to disclose communications made to him in official confidence when he considers that the public interests would suffer by the disclosure." (Sect. 124.) Medical men are not exempted from answering questions on matters which have come to their knowledge in professional confidence. A witness is not exempted from answering questions on the ground that his answers would tend to criminate himself, but, if compelled to answer, his answers cannot be used as evidence against him. (Sect. 132.)

In addition to the checks on the untruthfulness of a witness afforded by the provisions of the Penal Code relating to the giving of false evidence (sects. 191 to 195), and the publicity of judicial proceedings, there is the most effective one of requiring evidence to be given *vivâ voce*, in presence of the party against whom the evidence is produced, who has the opportunity of cross-examining the witness. Sects. 137 and 138 of the Indian Evidence Act provide that a witness shall be first examined by the party calling him. This is called examination-in-chief. He may then be "cross-examined" by the adverse party, and subsequently "re-examined" by the party calling him. In cross-examination "leading questions," *i.e.*, questions which suggest the answer wished for or expected (sect. 141), may be asked. (Sect. 143.)

Use of documents to refresh memory.—A witness while under examination may refresh his memory by the use of any writing, provided such writing was either

(1) Made by himself at the time of the transaction concerning which he is questioned, or so soon afterwards that the Court considers it likely that the transaction was at that time fresh in his memory; or

(2) Made by another person, and read by the witness, within the time aforesaid, if when he read it he knew it to be correct. (Sect. 159.)

A copy, with permission of the Court, may be used instead of the original document, provided the Court be satisfied that there is sufficient reason for the non-production of the original (sect. 159). Any writing used to refresh memory must be shown to the adverse party if required. (Sect. 161.) A witness may testify to facts mentioned in his notes, although he has no specific recollection of the facts themselves, if he is sure that the facts were correctly recorded in his notes. (Sect. 160.)

An expert may, while under examination, refresh his memory by reference to **professional treatises.** (Sect. 159.)

V. Presumptions, &c.—The Indian Evidence Act also contains various provisions relating to documentary evidence, admissions and confessions, the burden of proof, &c. Certain of the sections relating to the burden of proof will be referred to further on, under the subjects of presumption of death, and presumption of legitimacy. Here, however, it may be pointed out that in certain cases the law directs that when a certain fact, or set of facts, is proved to exist, a certain other fact shall as a consequence be presumed to exist. In such a case a presumption of the existence of this other fact is said to arise.

In certain cases the law directs that, certain facts being proved, no evidence shall be admitted in contradiction of the presumption arising therefrom. A presumption of this nature is called an **irrebuttable presumption.** Thus, according to the law of England (but not according to that of India) it is an irrebuttable presumption, that a boy under the age of fourteen is incapable of committing a rape. Hence, in England, if a boy is charged with this offence, and it is proved that he is under the age of fourteen, no evidence will be admitted to show that he was physically capable of committing the offence.

In other cases the law directs that certain facts being proved, the presumption arising therefrom may be negatived by proof of certain other facts. A presumption of this nature is called a **rebuttable presumption.** Thus, according to the law both of India and England, if a woman during the existence of a valid marriage between herself and a certain man, is delivered of a child, the child is presumed to be the legitimate child of the man to whom she is married. This presumption, however, is a rebuttable presumption, and may be negatived by proof that the parties to the marriage had no access to each other at any time when the child could have been begotten.

Hints for Guidance in Giving Evidence.

Recollect that your duties while in the witness-box may be of a twofold character: viz., (*a*) as an **ordinary witness**, to give evidence as to facts; and (*b*) as a **skilled witness** or expert, to give opinions. Be careful, therefore, in your evidence to distinguish between facts and opinions, and do not state as facts things which are merely matters of opinion.

In giving evidence as to facts, (1) **Bear in mind that oral evidence must be direct,** and confine your evidence as to facts, strictly to facts which you have yourself observed. If asked to give evidence as to a fact of which you have no personal knowledge, state that you have no personal knowledge thereof.

(2) **Be as precise as possible**; for example, be prepared with the date and time of each event about which you have to give evidence, with the exact measurement of wounds, the exact weight of powders and volume of fluids, &c., &c. You may be called upon to give evidence as to a statement made by an accused or dying person; if so, be prepared to state the exact words used (it is always advisable to note down these at the time). Recollect that no inducement, promise, or threat must be held out to an accused person in order to obtain a confession from him, and no leading questions should be asked of an accused or dying person.

(3) **Be careful to note all the facts** which it is your duty to observe. Thus, in making a post-mortem examination, do not neglect to examine and note the condition of every organ, so that you may be prepared to state not only what was, but also what was not, the cause of death.

In giving opinions (1) **Be careful not to overstep your legitimate duties** as an expert witness, by volunteering opinions on questions which are not questions of science or art. For example, the question whether certain characters you have observed in a wound indicate that the same was inflicted during life, involves a question of science or art, and it is part of your duty to, if called upon, give an opinion on this point. On the other hand, the question whether certain marks you have observed on the ground, or on various objects near the place where a dead body has been found, indicate that a struggle has taken place there, does not involve any question of science or art, and is not therefore a matter on which you should volunteer an opinion.

(2) **Consider with yourself what are the various points** on which you are likely to be called upon to give an opinion, *i.e.*, What inferences drawn from the facts would tend to support either side of the case. Consult the works of the leading authorities on these points; ascertain what opinions are therein expressed, and the grounds on which such opinions are held, and frame your own opinions with due regard thereto.

(3) **Be careful to draw no stronger inference,** and give no stronger opinion than the facts warrant you in doing. It frequently happens that the facts available do not justify a conclusive opinion being given one way or other. In such a case do not hesitate to state so, but be prepared to state precisely your reasons for being unable to give a definite opinion on the point.

In all cases (1) **Use the plainest language** consistent with accuracy; do not employ a technical term when an ordinary one will do.

(2) **If compelled to answer Yes or No** to a question which in your opinion does not admit of either answer being given without conveying a wrong impression, give that answer which most nearly approaches truth, but, having given it, request to be allowed to explain your answer.

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CHAPTER II.

GENERAL QUESTIONS CONNECTED WITH THE DEAD BODY, VIZ.:

I. HAS DEATH TAKEN PLACE, AND IF SO HOW LONG AGO? II. THE IDENTITY OF THE BODY. III. THE QUESTIONS OF PRESUMPTION OF DEATH AND OF SURVIVORSHIP. IV. THE CAUSE OF DEATH. V. THE DUTIES OF A MEDICAL MAN IN RELATION TO BODIES FOUND DEAD.

QUESTION I.—HAS DEATH TAKEN PLACE, AND IF SO, HOW LONG AGO?

The most reliable sign of death is the occurrence of **general putrefaction** of the body, but this **may be delayed** by various causes; *e.g.*:—

1. **Temperature.**—Below 32° F., putrefaction is entirely arrested. Above 32° F., and up to about 100° F., putrefaction is likely to be more rapid the higher the temperature. Temperatures above about 100° F. tend to delay putrefaction, and a temperature of 212° F. arrests it entirely.

2. **Non-access of Air.**—Free access of air promotes putrefaction, and *vice versâ*. Hence, protection of the body from access of air, by tightly-fitting clothes, enclosure in a tightly closed coffin, or complete submergence in water, tends to delay putrefaction.

3. **Absence of Moisture.**—Presence of moisture promotes, and absence of moisture retards, putrefaction. Hence, putrefaction is more rapid in moist than in dry air.

4. **Condition of the Body, and cause of Death.**—Putrefaction is more rapid in cases of sudden death, and in corpulent or dropsical bodies, than in cases of death from exhausting diseases and in emaciated bodies. The presence in the body of certain poisons, *e.g.*, arsenic, antimony, chloride of zinc, alcohol, chloroform, and phosphorus, tends to delay, and of others, *e.g.*, sulphuretted hydrogen and animal poisons, tends to promote, putrefaction.

5. **Application of antiseptics to the body.**—This, of course, retards putrefaction. Lime, contrary to the popular belief, retards putrefaction.

Instead of putrefaction the body may undergo—1. Mummification, in cold dry climates, or 2. **Saponification,** the soft parts undergoing conversion into **adipocere,** an ammoniacal soap. Saponification is said to be specially likely to occur in the case of bodies buried in deep moist graves, or wholly submerged in water.

The occurrence of general putrefaction of the body follows on the occurrence of complete molecular death, but complete molecular death does not occur immediately after somatic death, *i.e.*, death in the popular acceptation of the term. Hence we now have to consider what, **in the absence of putrefaction,** are the signs which indicate that death (*i.e.* somatic death) has taken place; these are—1. Cessation of circulation and respiration. 2. Loss of temperature of the body. 3. Supervention of cadaveric rigidity. 4. Minor signs.

I. Cessation of Circulation and Respiration.—That circulation has ceased cannot be inferred from a mere absence of the pulse. Auscultation even may fail to detect a very feeble action of the heart. Tidy[1] quotes a case of a man, *æt.* thirty-three, where for eight minutes no heart sounds could be detected, the man ultimately recovering. A few cases also are recorded of persons who have apparently possessed the power of voluntarily suspending the action of the heart (see *Cases* A 1 *a* and *b*). It may be pointed out as bearing on the question of the validity of cessation of circulation as a sign of death—

1. That the reliability of the test entirely depends on the accuracy of the means employed. And 2. That M. Rayer, from observations on the dying, assigned seven seconds as the maximum observed interval between the two last pulsations of the heart.

The cessation of respiration is ascertainable in various ways; *e.g.*, by holding a looking-glass to the mouth, absence of dimming of the glass indicates cessation of respiration. By the absence of movement in a feather or other light object held or suspended near the mouth or nostrils. By the absence of movement of a spot of light reflected from the surface of a shallow vessel of water or mercury placed on the chest. By careful auscultation. The movements of respiration may be so feeble as to be for all practical purposes imperceptible, but this condition cannot continue long without death taking place. In newly-born children, however, life has been known to continue for a considerable period, without respiratory movements being apparent. (See *Case* A 1 *c*.)

II. Loss of Temperature of the Body.—The normal temperature of the body (taken in the axilla) ranges between 97° and 100° F. After death the body tends gradually to acquire the temperature of the surrounding media, and if these, as is usually

[1] Legal Medicine, i. p. 138.

the case, are lower in temperature than the body, a gradual cooling of the body takes place. It is important, however, to recollect that in death from certain diseases the temperature of the body may, at the time of death, be higher than the normal, and may even rise considerably after death. Thus in cases of death from yellow fever, cholera, Bright's disease, abscess of the liver and other abdominal affections, rheumatic fever, small-pox, tetanus, and injuries to the nervous system generally, &c., a post-mortem rise of temperature, amounting in some cases to even 9° F., has been observed.[1] **Loss of temperature** of the body **may be delayed by**

(1) **Protection** of the body by non-conducting coverings.

(2) **High temperature** of the surrounding media.

(3) **Stillness** of the air surrounding the body.

(4) **Condition** of the body. The bodies of very fat persons cool slowly; those of children and old persons cool more rapidly than the bodies of adults.

III. Supervention of Cadaveric Rigidity (*Rigor mortis*) — After death the muscles commonly go through three stages, viz. (in order of their occurrence): 1. Relaxation. 2. Rigidity. 3. Relaxation.

In the first stage the muscles are relaxed, but contract on the application of stimuli. This stage in exceptional cases may last only a few minutes, or even be absent; it commonly, however, lasts about three hours. There is no case on record where this stage has lasted as long as twenty-four hours.

In the second stage, that of cadaveric rigidity or *rigor mortis*, the muscles become rigid, most probably from coagulation of myosin. Both involuntary and voluntary muscles are affected, and the rigidity occurs independently of nerve influence (paralysed limbs becoming rigid unless complete degeneration of the muscles has taken place), and independently also of the rate of cooling of the body. This stage commonly commences about three hours after death, and usually takes one to two hours to fully develop. Niderkorn found it to be fully developed before the end of the seventh hour after death, in 92 out of 113 cases.[2] The muscles become rigid most commonly in the following order, the limbs, &c., remaining fixed in the position existent at the time of the commencement of the rigidity. 1st. Those of the eyelids. 2nd. Those which raise the lower jaw. 3rd. Those of the face generally. 4th. Those of the limbs, usually upper and lower simultaneously, but sometimes the latter first. This stage lasts sixteen to twenty-four hours (Taylor), or, according to Tidy, twenty-four to forty-eight hours, and at its close the rigidity passes off in the same order as it came on, and the third stage commences.

[1] Tidy, Legal Medicine, i. p. 46.
[2] See Tidy's Legal Medicine, i. p. 62.

In the third stage the muscles are relaxed, but do not contract on the application of stimuli.

The duration of the two first stages greatly depends on the condition of the muscles at the time of death; if these are well developed, well nourished, and have not been subjected to great fatigue shortly before death, both stages are prolonged, and *vice versâ*. Thus in cases of death from exhausting diseases, the first stage may only last a few minutes, and the second only one or two hours. In cases also where just previous to death the muscles have undergone great fatigue, and also in cases where the irritability of the muscles has been exhausted by a powerful electric discharge, as in death from lightning-stroke, rigidity may come on at once, and the body stiffen in the position it was in at the time of death. On the other hand, in cases of sudden death, rigidity comes on late and lasts long, provided always, of course, that the muscles just previous to death have not been subject to great fatigue, or to anything tending to exhaust their irritability. Taylor and others apply the term "cadaveric spasm" to rigidity setting in immediately after death. Sometimes, it may be noted, important evidence as to the cause of death may be derived from the presence of matters grasped in the hands under the influence of cadaveric spasm (see *Case* A 14). Cadaveric rigidity may be distinguished from rigidity occurring during life: 1, by the low temperature of the body; and 2, by the fact that (provided the cadaveric rigidity is fully established) if a limb be forcibly bent, it does not resume its previous position.

IV. Minor Signs of Death.

1. **Changes in and about the eye,** *e.g.*, loss of sensibility of the pupil, loss of transparency of the cornea, loss of tension of the eyeball. None of these are reliable. The pupil may for a short time after death still respond to the action of atropia; and loss of transparency of the cornea, and of tension of the eyeball, may occur during life.

2. **Effect of heat upon the skin.**—A sufficient heat applied to the skin during life (or within a few minutes after death) produces—if the skin be not destroyed—true vesication, *i.e.*, vesicles containing serum rich in albumen. (See *Burns*, p. 94.)

Death may be simulated, to a certain extent, by certain diseased or abnormal conditions, *e.g.*, syncope, asphyxia, and catalepsy or trance. Also by the condition which, in very exceptional cases, individuals seem to possess the power of voluntarily assuming (see *Case* A 1 *a*, Colonel Townshend's case, and *Case* A 1 *b*). The suspended animation of newly-born children, again, may closely simulate death (see *Case* A 1 *c*). Death may be simulated for the purposes of extortion, &c.; but such cases are easily detected.

On the whole, the signs which may be relied on, as indicating that death has taken place, are—

1. The occurrence of general putrefaction of the body; or in the absence of this,

2 The absence of circulation and respiration for at least one hour, the gradual cooling of the body, and the gradual supervention of rigidity as the body cools.

After death the various changes which occur, viz cooling, rigidity, and putrefaction, **may be accelerated or retarded** by various conditions already enumerated. Paying due regard to these conditions, **an approximate estimate as to the period which has elapsed since death** may be formed from the progress these changes have made. The formation of such an estimate may be of great importance. The advanced state of putrefaction of a body may show, for example, that death must have occurred at a period considerably anterior to the date of disappearance of the individual whose body it is alleged to be; or as in *Case* A 2, the condition of the body of a murdered individual may show, that death must have taken place at a time when the accused had access to the victim and not subsequently to the time of termination of such access. The chief data available are as follows:—

1. **The temperature of the body.**—Observations (in temperate climates) show that the dead body cools down to a temperature a little above that of the external air, in fifteen to twenty hours, the fall of temperature being more rapid in the earlier than in the later hours after death.

2. **The condition of the muscles.**—If these are relaxed and contractile (*i.e.*, in the first stage), it is probable that not more than three to six hours has elapsed since death. If rigid (*i e*, in the second stage), it is probable that more than three and less than thirty-six hours has elapsed since death.

3. **The amount of putrefaction.**—Other things being equal, the greater the amount of this the longer the period which has elapsed since death. The rapidity of putrefaction, however, varies so greatly with circumstances that no general rule can be laid down for estimating by its amount the length of time which has elapsed since death occurred.

QUESTION II.—THE IDENTITY OF THE BODY.

The question of identity may arise in regard to the living, as well as the dead. Further, two of the factors whereby identity may be established, are the age and sex In order to avoid repetition, therefore, it is convenient to consider in this place the whole subject of identity, including age and sex, of the living as well as of the dead

As regards **identity of the dead**, it may be remarked, that it is of the utmost importance, especially in India, to note when examining a dead body, everything that may lead to its identifi-

cation. Dead bodies can be obtained with comparative ease in India, and this affords special facilities for the fabrication of evidence in support of false charges of murder. Thus, in *Cases* A 3, convictions for murder were obtained by the production of bodies, falsely alleged to be those of individuals who had disappeared. In *Case* A 3 *b*, the prisoners even confessed to the murder; and yet in both cases the individuals supposed to have been murdered were, after conviction of the alleged murderers, produced alive in Court. Sometimes fragments only of a body are found, here also it is of the utmost importance to note everything which may lead to their identification (see *Cases* A 4).

Medical expert evidence may also be required in cases where the question at issue is the **identity of a living individual**. Two celebrated cases of this kind, turning entirely on the question of the identity or otherwise of a living person, with an individual who had not been heard of for years, are the Tichborne case, and the case of Martin Guerre (*Cases* A 5 *a* and *b*).

Identity may be established by proof of the existence of certain personal peculiarities. These may conveniently be considered under two heads, viz. (1) **Congenital peculiarities**, and (2) **Acquired peculiarities.**

Congenital peculiarities.—These may, or may not, be hereditary, but it must be recollected that a hereditary peculiarity sometimes misses one generation, appearing in the next (*atavism*). Under the head of congenital peculiarities may be mentioned (1) **Sex** (see this subject). (2) **General character of the features** and resemblance of these to parents, or to photographic or other portraits (see *Case* A 5 *a*). In the case of dead bodies, putrefaction rapidly renders the features unrecognizable; in some instances, however, the features have been clearly recognized after long interment, *e.g.*, in the case of Charles I., whose body was exhumed 165 years after death. (3) **Colour of the eyes.** In some individuals one iris differs in colour from the other. (4) **Colour of the hair.** The hair resists putrefaction, hence its colour, &c., may be of special importance in the case of exhumed or greatly putrefied bodies. The colour of the hair may, however, have been altered, *e.g.*, darkened, generally by the use of metallic dyes, chiefly lead or silver compounds;[1] or rendered lighter by chlorine or hydrogen dioxide solution, in which case the roots will be found darker than the rest of the hair. (5) **Presence of deformities**, such as hare lip, hypospadias, additional digits, web fingers or toes, or of marks on the skin, such as moles or nævi. (6) **Tendency to heredi-**

[1] For the detection of these the hair may be digested in dilute nitric acid, the acid liquid evaporated to dryness, and the usual tests applied to a solution of the residue; or the hair may be incinerated, and the metal sought for in the ash (see detection of lead in organic mixtures, p. 181).

tary diseases, *e.g.*, gout.[1] (7) **Peculiarities of voice, or of tricks of manner.** As an example of heredity in the latter, Woodman and Tidy mention the case of a man who never knew or saw his father, but who, like him, shot from the left shoulder.[2]

Acquired Peculiarities.—(1) **Age** (see this subject). (2) **The height.** This may have to be determined from the measurement of one or more detached bones (see Professor Humphreys' Table, *Appendix*, p. 493). (3) **General condition** of the body, whether fat, or well nourished and healthy, or muscular, or the reverse. (4) **Traces left by old injuries,** such as fractures, wounds, or burns. An old fracture may enable human remains to be identified long after entire disappearance of the soft parts. **Cicatrices,** except from very slight wounds, are permanent. The scars of very slight wounds, not involving loss of substance, may, or may not, in time disappear. Cicatrices tend to contract, and become more linear, with lapse of time. They are always less vascular than the rest of the skin, hence friction renders them more visible. They never contain sebaceous or hair follicles. (5) **Tattoo marks.** These are in general permanent, but in a small proportion of cases, especially if a non-carbonaceous pigment has been employed, they disappear after several years. Tattoo marks may be got rid of by escharotics, but in such cases a scar will be left, and, on friction, traces of the tattooing, in the form of fine white lines, often become visible. Strong friction also, will render a faded tattoo mark more distinct. (6) **The teeth and hair.** Loss or deformity of the teeth, denudation of hair from the scalp or other parts of the body, presence of false teeth, or correspondence of the formation of the jaw with a mould taken for the purpose of fitting artificial teeth, may all be of great importance in establishing identity (see *Case* A 4 *a*). (7) **Recollection of past events.** This is often of great importance in cases of imposture (see *Case* A 5 *a*). (8) **Marks due to religious or other customs,** or owing their origin to the occupation of the individual. Under this head come (*a*) Circumcision; (*b*) Perforations in the ears. The position of these should be noted, whether through the lobes only, or in other situations, and whether both or only one ear is pierced. Perforation of the **nasal septum,** or alæ, should also be looked for. (*c*) **Brown marks** on the skin, **due to constant exposure** of particular portions to the action of the sun. On the bodies of natives of India such marks should be specially looked for on the front of the trunk, and dorsum of the foot. On a male body a narrow linear, nearly vertical mark, may be found on the front of the chest, either on the right side (in a Hindu), or on the left side (in a Mahomedan). On a female body the front of the chest, usually covered by a bodice, may be found paler than the rest of the skin, and peculiarities in shape of the bodice may be

[1] See also Life Assurance, p. 386. [2] Forensic Med. p. 661.

traceable by noting the outline of this paler portion. Again, if the individual has been in the habit of wearing shoes without stockings, the dorsum of the foot, down to the metatarsal bones, may be found darker than the rest of the foot. (*d*) **Callosities** on the skin of the hands may be found, indicating laborious occupation, or on various parts of the body, indicating habitual assumption of a particular posture. Chevers[1] points out that such callosities, arising from the posture assumed in praying, will usually be found in certain situations on the bodies of devout Mahomedan males, *e.g.*, on the centre of the forehead, the tip of the left external malleolus, and on the outer side and dorsum of the left foot, &c. Under this head may also be mentioned stains of henna on the hands or feet, marks of red or white paint on the forehead or other parts of the body, and marks of tattooing between the eyes or on the wrists.

The clothes or ornaments found on a body may aid in establishing its identity. In the case of natives of India, the following points should be specially noted. **In males** (1), if the native **coat** (*Angarka* or *Chapkan*) is worn, whether this fastens on the right side (= Hindu), left side (= Mahomedan), or centre (= Parsees, Jews, and some Hindus). (2) If a **sacred thread** is worn, whether this passes over the left shoulder and under the right armpit (= Hindu), or is worn round the waist (= Parsee). (3) If a **necklace** of beads is worn, the material of which they are composed should be noted. If these are of wood, or if the beads are nuts or seeds, the wearer is probably a Hindu. It may be also noted that unless both ears are pierced, the individual is not a Hindu. In the case of **females** it should be noted (1) whether **trousers** are worn or not; trousers with a sacred thread indicate a Parsee; without, a Mahomedan. (2) Whether there are **bangles on the wrists** or not. An adult female without bangles is probably a Hindu widow, or a non-Hindu. (3) Whether the **nose ring** is passed through a perforation in the septum (= Mahomedan) or through one in the left ala (= Hindu). (4) Whether the **head is shaved** or not; a female with the head shaved is probably a Hindu widow.

Amount of light sufficient for identification.

(1) **A flash of lightning** undoubtedly affords sufficient light to enable an individual to so distinctly discern the features, &c., of another, as to be able to subsequently recognise him.

(2) **The flash caused by the discharge of a pistol or gun,** provided the circumstances are favourable, also similarly affords sufficient light for recognition and identification. Favouring circumstances are: close proximity to the discharge on one side

[1] Med. Jurisp., p. 58.

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of the line of fire; absence of other light; and not much smoke from the powder.

(3) **Flashes of light from blows on the head** being merely subjective sensations, cause no illumination of external objects, hence recognition and identification thereby is not possible.

Age.

Determination of age may be required for purposes of identification, and also when the question is one of

(1) **Criminal responsibility.**—Children under the age of seven are deemed incapable of committing an offence. Children between the ages of seven and twelve in India (seven and fourteen in England) are only deemed capable of committing offences if they have attained a certain degree of maturity of understanding.[1] Sexual intercourse with a girl under the age of ten is (in India) **rape**, even if the girl consents.[2] In England, sexual intercourse with consent is rape up to the age of thirteen, and between the ages of thirteen and sixteen, although not rape, is an offence.[3] Only a person over the age of twelve can give **a valid consent** to suffer any harm which may result from an act done in good faith, and for the sufferer's benefit (Penal Code, sect. 90); and in cases where the act does not come within this description, the consenting individual must be of the age of eighteen or more, for his consent to be valid (Penal Code, sect. 87).

(2) **Eligibility for certain kinds of employment.**—By law in England, in factories children under eight may not be employed, and children between eight and thirteen may only be employed for six and a half hours per day. Further, males between thirteen and eighteen, and females, may only be employed for sixty hours per week. The Indian Factories Act (XV. of 1881), provides that, in factories coming under its operation, no child under the age of seven shall be employed, and that children between the ages of seven and twelve shall not be employed for more than nine hours per day, and shall have one hour daily for rest, and four unemployed days per month.

(3) **Capacity to contract marriage.**—According to the law of England, females under the age of twelve, and males under the age of fourteen, cannot contract marriage.

(4) **Attainment of majority.**—In England majority is attained at twenty-one. Persons under this age are minors. A minor cannot make a valid will, cannot alienate his goods by deed,

[1] See *ante*, p. 2. (Penal Code, sects. 82 and 83).

[2] Penal Code, sect. 375, see *post*, p. 319.

[3] Criminal Law Amendment Act, 1885 (48 and 49 Vict. c. 69). Sect. 7 of this Act also makes it an offence to abduct an unmarried girl under eighteen with intent that she should be unlawfully and carnally known by any man.

cannot be called upon to serve on a jury, &c. Certain cases excepted, persons domiciled in British India attain majority on completion of their eighteenth year.

Legally an individual attains a given age on the first minute of the day before his birthday, *e.g.*, an individual in England will, say, popularly speaking, be twenty-one on the 3rd of May, legally his minority ceases at the end of the last minute of the 1st of May.

The principal data available for the determination of age are—1, the teeth; 2, the height and weight; and 3, the progress of ossification.

(1) **The teeth.** The following tables show the probable times of eruption of (*a*) the temporary, and (*b*) the permanent teeth.

(*a*) THE TEMPORARY OR MILK TEETH.

Order in Jaw, from centre.	Name of Tooth.	Probable Age of eruption in months.	
		Bell.	Guy.
1	Incisors, central	5–8	5–7
2	„ lateral	7–10	6–9
3	Canines	14–20	15–18
4	Molars, anterior	12–16	8–15
5	„ posterior	18–36	18–24

(*b*) THE PERMANENT TEETH.

Order in Jaw, from centre.	Name of Tooth.	Probable Age of eruption in years (Saunders).
1	Incisors, central	8
2	„ lateral	9
3	Canines	12–12½
4	Bicuspids, anterior	10
5	„ posterior	11
6	Molars, anterior	7
7	„ second	12½–14
8	„ posterior	18–25

Between the sixth and seventh year the jaws may contain as many as forty-eight teeth, viz., twenty perfect temporary, and twenty-eight more or less developed permanent teeth.

(2) **The height and weight.**—According to Harvey, the average weight of Indian children at birth is 5½ lbs. According

to Tidy, English children at birth average 6½ lbs. in weight, and during the first year after birth gain in weight at about the rate of 1 lb. per month. In the Appendix will be found Quetelet's table[1]—calculated from observations on Belgians—of average heights and weights year by year from birth up to thirty. According to this table, a boy attains about half his adult weight at the age of thirteen, and a girl about half her adult weight at twelve, or rather under. Dr. Bridge's observations on English children[2] give as the age of attainment of half adult weight, in boys about twelve, and in girls eleven, or rather under. The height and weight of adults of the majority of the races inhabiting India is, it should be noted, lower than that of Europeans.[3]

(3) **The progress of ossification.**—In the Appendix[4] will be found Ogston's table of the development of the skeleton; here it may be noted that—

a'. **Points of ossification** appear in the following **epiphyses** at the ages stated (in years). Before the end of the second, in the heads and lower epiphyses of the humerus, femur, and tibia, and in the lower epiphyses of the ulna, radius, and fibula. At five in the upper epiphysis of the fibula, at seven to nine in the olecranon, and eight to ten in the upper epiphysis of the radius.

b'. **Points of ossification** appear in the **bodies** of the following bones at the ages stated (in years). At two and a half, patella; at three, cuboid; at four, trapezoid, and second and third tarsal cuneiform; at five, semilunar and carpal scaphoid; and at twelve, in the pisiform bones.

c'. **Bony union takes place** at one year, of the posterior arches with the bodies of the vertebræ, and of the three portions of the temporal bone. At three years, of the odontoid process with the axis. At four years, of the styloid process with the temporal bone. At six years, of the ascending and descending rami of the pubis. At nine years, of the three portions of the os innominatum in the acetabulum. At fifteen years, of the last four sacral vertebræ, and of the coracoid with the scapula. At about twenty-five, all the epiphyses have united; and at twenty-five to thirty, the first sacral vertebra unites with the others.

d'. As age advances, the rib and laryngeal cartilages become ossified, and the skull becomes thinned by absorption of diploe. The angle of the jaw—obtuse in infants, and nearly a right angle in adults—becomes obtuse again in old age, and in old age the alveolar processes of the jaws become absorbed.

Sex.

Determination of sex may be required for purposes of identification, and also when the question is one of

[1] Appendix, Table III., p. 494.
[2] *Ibid.*
[3] *Ibid.*, Table II., p. 493.
[4] *Ibid.*, Table IV., p. 494.

1. **Right of succession to property.**
2. **Right of vote at elections.**
3. **The manner in which the individual is to be educated.**
4. **The manner in which the individual is to enter on the contract of marriage.**

When the soft parts are entire, there will, of course, in the great majority of cases, be no difficulty in determining sex. In exceptional cases, however, the sex may be doubtful. Such cases may be conveniently divided into three classes, viz.:—

1. **Androgynæ**, or manly women; *i.e.*, cases in which the individual, really a female, to a certain extent resembles a male. 2. **Androgyni**, or womanly men; *i.e.*, cases in which the individual, really a male, to a certain extent resembles a female. And 3. **Hermaphrodites**; *i.e.*, individuals in whom the two sexes are in some sort blended.

I. Androgynæ.—In these the resemblance to the male organs is almost always due to an enlarged clitoris. The **clitoris is distinguished from the penis** (*a'*) by the absence of the corpus spongiosum; (*b'*) by its being always imperforate; and (*c'*) by its being, even in cases of greatest enlargement, always smaller than the penis. A prolapsed uterus has been mistaken for a penis; such a condition would, however, hardly deceive a medical man. It may be noted, however, that the fissure of a prolapsed uterus is transverse, while that of the penis is vertical.

II. Androgyni.—In these cases the abnormal condition of the sexual organs is usually due to arrest of development. The most usual condition in such cases is as follows:—The individual has a small imperforate penis, with a vertical slit beneath, which may lead into a canal of some size. The scrotum is cleft in two by the slit, the halves bearing some resemblance to the labia. The portions of the divided scrotum may each contain a testicle, or one or both of these may be absent from non-descent. The testicles are usually small. The canal beneath the penis is short, opens into the bladder, and shows the parts found at the base of the urethra, viz., the veru montanum, sinus pocularis, and seminal orifices. There are no nymphæ, and there is no hymen. Such cases may, in fact, be regarded as an intense form of hypospadias, this term being applied to all cases where the penis is split below.

III. Hermaphrodites.—Each sexual organ in the one sex has its corresponding analogue in the other; thus—

Male		Female.
The Penis	corresponds to	The Clitoris.
The Prepuce	"	The Nymphæ.
The Scrotum	"	The Labia.
The Sinus Pocularis	"	The Uterus.
The Vasa Deferentia	"	The Fallopian Tubes.
The Testicles	"	The Ovaries.

The old idea of a hermaphrodite was, a person with a complete set of sexual organs of the one sex plus a complete set of the sexual organs of the other sex, and capable of self-impregnation. No such individual has ever been known to exist. Cases, however, have occurred where in the same individual certain organs of the one sex have been found accompanied by certain of the organs of the other; as, for example, a testicle on one side and an ovary on the other (lateral hermaphroditism); or the external genital organs may be male, and the internal female (transverse hermaphroditism). In such cases the sex may be very difficult to determine. No definite rules can be laid down on the subject; each case must be decided on its own merits, following the legal rule, viz., that the individual is to be considered to be of that sex which most prevails.

In cases of doubtful sex, in addition to the appearances presented by the organs of generation, the **direction of the sexual desires** of the individual should be inquired into, and note should be taken of the **general configuration** of the body. The following are the chief points in which the general configuration of the body differs in the two sexes:—

1. In the male the **shoulders** are generally wider than the hips; in the female, the reverse is the case.

2. The **sexual hairs** on the pubes extend higher towards the umbilicus in the male, than in the female.

3. In the male the **umbilicus** is nearer to the pubes than to the scrobiculus cordis; in the female the reverse.

4. In the male the **voice** is deeper, and there is more hair on the face than in the female. In the female the **breasts** are more developed than in the male.

On the whole, in cases of doubtful sex,

1. The presence of a **testicle** or testicles, accompanied by emissions of a fluid containing spermatozoa, is the strongest possible evidence of the individual being a male.

2. The presence of an **ovary** or ovaries, accompanied by periodic hæmorrhages from an opening about the genitals, is very strong evidence of the individual being a female.

3. In the absence of the above characters, the presence of a **uterus**, or of a second opening distinct from and behind that leading into the bladder, indicates a female.

4. If the general configuration of the body agrees with the indications derived as above from examination of the sexual organs, this may be looked upon as confirmatory evidence, but if it disagrees it should be disregarded.

In the Appendix will be found an abstract of the case of Levi Suydam, and of other instructive cases of doubtful sex (*Cases* A 6).

It should also be noted that cases of concealed sex occasionally occur. A remarkable instance of this class is the

case of Dr. James Barry, an army surgeon, who rose to the rank of Inspector-General of Hospitals, and, after death, was discovered to be a female.

If the soft parts are destroyed, and the skeleton alone is available, the following characters distinguish the bones of a female from those of a male:—

1. The **bones** are thinner, lighter, less curved, and the muscular ridges are less marked, than those of a male.

2. The female **skull** is smaller, more oblong, and the chin is less prominent than in the male.

3. The **chest** is deeper, the sternum shorter, the ribs thinner, the rib cartilages longer, and the clavicles thinner and straighter than those of a male.

4. The **pelvis.**—In the female the ilia are more expanded, the sacrum more concave, the symphysis shallower, the arch larger and its edges more everted, and the foramina more triangular, than in the male. In the female also the pelvis is wider, and its outlets larger. (For measurements of the male and female pelvis, see *Table* V., *Appendix*, p. 495.)

QUESTION III.—THE PRESUMPTION OF DEATH AND OF SURVIVORSHIP.

1. **The Question of the Presumption of Death.**—When will it be presumed that a person is dead? In India the law is (*a*) that if a person is proved to have been alive within thirty years, the legal presumption is that he is still alive, except (*b*) it is proved that the person has not been heard of for seven years by those who would naturally have heard of him if he had been alive, in which case the law presumes that he is dead (sects. 107 and 108 Indian Evidence Act). The law, however, presumes nothing as to the time of his death, the period of which, if material (as it often must be in cases of succession and inheritance), must be proved by evidence. In either case, the presumption arising may be rebutted by proof, in case (*a*) of the person's death; in case (*b*) of his being still alive. In France, a legal presumption of death arises after thirty-five years of absence, or after one hundred years from date of birth.

2. **The Question of Presumption of Survivorship.**—When two or more persons die at almost the same time, or by a common accident, the question may arise who survived longest; and if no direct evidence on this point is available the question becomes one of presumption of survivorship. As an example of the cases in which this question arises, suppose A to have left property by will to B, and that A and B die by a common accident, no direct evidence being available as to whether A or B died first. Here the question of presumption of survivorship may arise, because if A died before B, B may be considered to have suc-

ceeded to the property left him by A, and B's heirs inherit; while if B died first, A's heirs inherit, seeing that B never succeeded to the property willed to him by A. In some countries definite rules of law exist by which such cases are decided. In France, for example, some of the rules laid down are: (1) If all those who perished together were under fifteen, the oldest shall be presumed to be the survivor. (2) If all were over sixty, the youngest shall be presumed the survivor. (3) If all were between fifteen and sixty, the males shall be presumed to have been the survivors if the ages were equal, or the difference in age not greater than one year; in other cases the youngest shall be presumed the survivor. The English law presumes nothing in cases of this kind, and if therefore a person made a claim and had, in order to substantiate it, to prove that A survived B, and had no proof of that fact beyond the assumptions arising from age or sex, he could not succeed. It may, however, be pointed out, that in questions of this kind it is likely that the strongest lived longest. There are, however, certain exceptions; *e.g.*,

(1) When a mother and child both die during delivery, if the death of the mother has been caused by hæmorrhage, it is probable that the mother died first. (2) If a number of persons die from the effect of excessive heat, it is probable that the adults died first, children and old persons bearing heat better than adults. (3) When the cause of death is drowning, as females are more likely to faint than males, and as the occurrence of syncope delays death by asphyxia, it is possible that females may survive longer than males. If, however, there has been a struggle for life, it is probable that the males, being stronger, survived the females. (4) Where the cause of death is starvation, aged persons (if healthy and robust) requiring less food than adults and children, probably live longest.

QUESTION IV.—WHAT WAS THE CAUSE OF DEATH?

At present only certain general points bearing on this question can be considered, viz.:—1. The modes of death; and 2. The relative frequency of occurrence of the chief forms of death by violence.

I. The Modes of Death.

Bichat classifies the modes of sudden death as follows:—(1) Death beginning at the heart, or **syncope**. (2) Death beginning at the head, or **coma**. (3) Death beginning at the lungs, **asphyxia or apnœa**. As to the causes of these—

I. Syncope. This may be divided into *a'*, **anæmia**, arising from deficient supply of blood, and *b'*, **asthenia**, arising from deficient power of the heart muscle. Syncope may arise from

1. **Deficient supply of blood,** as in death from hæmorrhage.

2. **Altered quality of the blood,** as in death from the action of certain poisons, *e.g.*, digitalis.

3. **Disease or degeneration** of the heart muscle, *e.g.*, fatty degeneration.

4. **Shock,** as in death from lightning-stroke.

II. Coma may arise from

1. **Deficient supply of blood,** *e.g.*, from the plugging of an artery supplying the brain.

2. **Altered quality of the blood,** as in narcotic poisoning.

3. **Pressure on the brain,** of depressed bone, blood, products of inflammation, or morbid growths.

III. Asphyxia or apnœa may arise from

1. **Cessation of the respiratory movements,** owing to (*a*) **Paralysis** or exhaustion of the muscles of respiration. (*b*) **Tetanic spasm** of the muscles of respiration. (*c*) **Mechanical pressure** on the chest.

2. **Non-expansion of the lungs** owing to (*a*) Presence of **air or liquid** in the **pleura.** (*b*) **Division or compression** of the **pneumogastric.**

3. **Exclusion of oxygen** from the lungs, partial or complete, owing to (*a*) **Rarefaction** of the respired **air.** (*b*) **Admixture of inert gases** with the respired air. (*c*) **Mechanical obstruction** of the air passages by impaction of foreign bodies, submersion, presence of a ligature, spasm of the glottis, &c. (*d*) **Disease** of the lungs, *e.g.*, hepatization.

4. **Exclusion of blood from the lungs,** as from plugging of the pulmonary artery by a clot (embolism).

The prominent symptoms of each mode are :—

In Syncope.—Pallor, coldness of the surface. Pulse feeble, slow and irregular or fluttering (in anæmia), or frequent (in asthenia). Gasping respiration, followed in anæmia by insensibility and convulsions.

In Coma.—Stupor, deepening into complete insensibility. Stertorous breathing, slow laboured pulse. In this mode of death unconsciousness precedes difficulty of breathing.

In Apnœa.—Intense struggle for breath, giddiness followed by unconsciousness and convulsions. In this mode of death, difficulty of breathing precedes unconsciousness.

The chief post-mortem appearances of each mode are :—

In syncope, if death has occurred by anæmia both sides of the heart are found empty ; and the heart itself, if the examination be made soon after death, is found contracted. If death has occurred by asthenia the heart is found relaxed, with its cavities empty or full ; if the latter, both sides are equally full.

In both coma and apnœa, the right side of the heart is found full of blood, and the left side empty. In both there is

engorgement of the lungs, but this is much greater in amount in apnœa than in coma. In apnœa there is also great general venous engorgement.

II. The relative frequency of occurrence of the chief forms of death by violence.

(*See Tables VI. to IX., Appendix, p.* 496.)

It may first be pointed out that in England the average annual death-rate from all causes is about 22·4 per 1000 of population, and that of the total deaths occurring yearly about 3 per cent. (or about ·70 per 1000 of population) are due to violence. Indian mortality statistics are not very reliable, but from such as are available it would appear, that while in India the annual death-rate from all causes is higher than in England, the death-rate from violence is lower (·36 to ·52 per 1000 of population). It is noticeable, also, that while in England about three times as many males as females die annually from violence, in India the death-rate from violence of the two sexes is nearly equal. Both in England and in India, of the deaths from violence by far the largest number are due to accident, suicide ranking next in order of frequency, and homicide last.

Accident.—In England about 87·5 per cent. of the deaths from violence (= about ·6 per 1000 of population) are due to accident, the male death-rate from accidental violence being rather more than three times as great as the corresponding female rate. In India, as far as can be gathered from published statistics, the death-rate from accidental violence equals about ·3 to ·4 per 1000 of population, the male rate in most provinces slightly exceeding the female rate. In India the most common causes of death from accidental violence are drowning, snake bite, and injuries inflicted by wild animals. In the Bombay Presidency, for example, in 1883, accidental drowning accounted for about one-third of the total deaths from violence of the year; and in most Indian provinces about one-fourth to one-third of the violent deaths occurring yearly are reported as due to snake bite and wild beasts.

Suicide.—Annual Rate. Just under one-tenth of the violent deaths reported yearly in England (about 65·2 per million of population) are cases of suicide. Bengal and the Punjaub excepted,[1] the reported annual death-rate from suicide in the different Indian provinces ranges from about 50 to 80 per million of population. A remarkable difference between English and Indian suicide statistics is, that while in England the suicide

[1] "In these two provinces the reported average annual suicide rate, for the five years ending 1876, was, Bengal 31·3, Punjaub 12·7 per million of population."—K. McLeod, 'On Suicide in India.'

death-rate among males is very nearly three times as high as among females, in the different Indian provinces the female suicide rate exceeds the corresponding male rate. Thus in the Madras Presidency, where the rates for the two sexes differ least, the female suicide rate is about one-tenth higher than the male rate; while in the North-West Provinces, where the rates differ most, the female suicide death-rate is on an average about two and a half times as high as the male rate. **Suicide by children** is not uncommon in India. In Bengal, for example, in 1872, of 1716 suicides 23 were children; and in Oudh, in the seven years ending 1876, of 4172 suicides 46 were children. **The modes of suicide** most commonly employed in India are drowning, hanging, and poison. Drowning is the mode selected by about three-fourths of the female suicides of the Madras and Bombay Presidencies, while more than three-fourths of the male suicides in the same provinces hang or drown themselves in about equal numbers. In the Punjaub one-half the male, and nearly one-half of the female suicides choose hanging, while drowning is selected by only about one-third of the females and one-sixth of the males. Hanging, also, is the mode chosen by over half of the female and about one-third of the male suicides of Calcutta. Poison, usually arsenic or opium, is chiefly used as a means of suicide in certain special localities, *e.g.*, in districts where the poppy is grown, and in the towns of Calcutta and Bombay. **Of the causes leading to suicide** in India, the following deserve special mention, either from the frequency with which they give rise to cases, or on account of their peculiar character. (1) **Grief or shame.** This is a frequent cause of suicide in India. Numerous instances are recorded of suicide by wives after quarrels, sometimes trifling in character, with their husbands, or with their husbands' relatives. Pregnancy following illicit intercourse—a not uncommon result of enforced widowhood—has also in many recorded cases led to suicide from shame and distress, and even to homicide (see *Cases* C 23 to 26). In the case of males, more or less common causes of mental distress leading to suicide are, domestic quarrels and pecuniary losses. Instances are also met with of suicide from distress of mind arising from arrest on criminal charges. (2) **Physical suffering.** Chevers, McLeod, and others, notice that severe physical, especially abdominal, pain, is in India a frequent more or less direct cause of suicide, particularly among females. (3) **Revenge.** In India cases are sometimes met with in which an individual who has been injured by another kills himself, under the idea that he thereby throws the responsibility for his death on the person who has injured him. Instances quoted by Chevers show that, under the name of "*chándi*," this form of a suicide was a well-known custom among the ancient Rajpoots. A variety of this description of suicide is the practice known as sitting "*dharna*,"

or starving oneself at the door of an enemy or debtor. Sometimes a suicide from revenge endeavours to destroy himself in such a manner as to raise a suspicion of homicide against his enemy. (4) **Religion.** Self-destruction from religious motives was formerly of somewhat frequent occurrence in India. One variety of this form of suicide consisted in the individual offering himself as a sacrifice, in order to propitiate one of the Hindoo deities, as, for example, by casting himself under the wheels of the car of Juggernauth, or drowning himself in the Ganges. No doubt, also, in some cases of "*sati*," or burning of widows on the funeral pile of their husbands, formerly of frequent occurrence in India, the victim was a consenting party.

Homicide.—In England, about 3 per cent. of the total deaths from violence occurring yearly are reported as homicidal cases, equal to a death-rate from homicide of 21·6 per million of population. Reliable statistics as to the death-rate from homicide in India are not available, but in the Bombay Presidency, in 1875, the number of violent deaths classed as "otherwise," *i.e.*, not known to be accidental or suicidal, equalled 26·19 per million of population. Of these (see *Table IX*) more than one-half were deaths from drowning, and nearly one-half deaths from wounds. Some of the more common or remarkable forms of homicide in India may now be briefly mentioned. (1) **Homicide connected with the relations between the sexes.**—Under this head may be mentioned as more or less common in India (*a*) Murders of husbands by their wives. Here the motive is usually either revenge for ill-treatment, or the facilitation of an intrigue, and very frequently poison—often in the latter class of cases supplied by the paramour—is the means resorted to. Possibly, however, in some cases of husband poisoning, the poison is given as an aphrodisiac, and not with homicidal intent.[1] (*b*) Murder by way of punishment for adultery. Here very often mutilation of the body of the victim accompanies the murder. In India, in fact, mutilation of the nose, ears, lips, &c., is a very commonly resorted to method of punishing a woman for sexual infidelity. (*c*) Murder of females pregnant from illicit intercourse (see *Cases* C 25 and 26). In cases of this description the victim is frequently a Hindu widow, and very often the fatal result is a consequence of injuries inflicted for the purpose of procuring abortion. (*d*) Murder of females after violation. Many cases are on record in India of this description of homicide, the victim in some instances being a young girl, in others an adult female; in the latter class of cases usually more than one man is concerned in the crime. (2) **Homicide connected with acquisition.** More or less common examples of this are (*a*) Homicide arising out of disputes in regard to the possession of land. Often such disputes lead to affrays, in which clubs and other blunt weapons are freely used

[1] See Arsenic, p. 148, and case B 16*a*.

with fatal results. (*b*) Death from injuries inflicted by gang robbers or dacoits,[1] the injury being sometimes inflicted by way of torture, often by burning, in order to extort information as to the place in which money or valuables have been hidden. (*c*) Murder of young children for the sake of the ornaments worn by them. This is a variety of homicide of tolerably frequent occurrence in India. *Cases* A 35 and 36 are examples. (*d*) Thuggee[2] or road robbery accompanied by homicide. This description of murder used formerly to be often met with in India, strangulation being the means commonly employed. Thuggee, however, is now rare, and in such cases as now occur, the death of the victim is usually the result of drugging, dhatura being the agent commonly used. (*e*) Murder by way of punishment for theft. Cases are not infrequently met with in India in which thieves caught in the act are set upon and violently beaten, perhaps killed, mutilation—*e.g.*, severance of the right hand, or of the ears—often accompanying the murder. (3) **Homicide in which the victim or victims are unoffending persons.** Murder cases often occur in India in which the victims are numerous, and include children or others who have given the murderer no offence. In cases of arsenical poisoning, for example, the victims are often several in number, some being children (see *Cases* B 14 *c* and 15 *b*); and often in such cases the injury to avenge which the murder is committed is of a very trifling character. Again, in running amok cases, it frequently happens that some or all of the victims are unoffending persons (see *Cases* B 36 and 38 *a* and *b*). Cases also are sometimes met with in India in which an individual, in order to revenge himself on an enemy, kills some unoffending person, sometimes a relation or friend, solely for the purpose of bringing a false charge of murder against the person who has injured him (see *Case* A 10). (4) **Homicide with consent of the victim.** In India it sometimes happens in a case of homicide that the individual killed has consented to suffer death. Thus, for example, in the cases of homicide, for accusation just referred to, the victim is sometimes a consenting party to the crime. The custom of the burying alive—"*samadh*"—of lepers, which formerly was widely prevalent in India, affords another example of this description of homicide, as, at any rate in the great majority of cases, the sufferer used to be a consenting party. (5) **Homicide connected with superstition.** Human

[1] "Where five or more persons conjointly commit, or attempt to commit, a robbery, or where the whole number of persons conjointly committing, or attempting to commit, a robbery, and persons present and aiding such commission or attempt amount to five or more, every person so committing, attempting, or aiding, is said to commit dacoity." (Indian Penal Code, sect. 391.)

[2] "Whoever at any time after the passing of this Act shall have been habitually associated with any other, or others, for the purpose of committing robbery, or child-stealing, by means of or accompanied with murder, is a Thug." (Indian Penal Code, sect. 310.)

sacrifice as a religious rite, several cases of which are mentioned by Chevers, formerly widely prevailed in India, but has now been, probably entirely, suppressed. The same may be said of the practice of "*sati*," or widow burning, before alluded to, and of the practice of burying widows alive in their husbands' graves, formerly prevalent among certain castes. Cases of homicide connected with superstition still, however, occasionally occur in India; *e.g.*, the killing of individuals suspected of witchcraft, and cases in which death results from the subjection of the victim to an ordeal for the discovery of theft (see *Case* B 16 *c*), or of supposed practice of witchcraft (see *Drowning*, p. 79). (6) **Murder of infants.**—For the peculiar features of this crime in India, see *Infanticide*, p. 351.

QUESTION V.—THE DUTIES OF MEDICAL MEN IN RELATION TO THE DEAD BODY.

These may include:—

I. Examination of the position and attitude of the body, and of its surroundings.

II. External examination of the body, including examination of its coverings.

III. Internal examination of the body.

I. Examination of the position and attitude of the body, and of its surroundings.

If summoned to the spot where a dead body has been found, and is still lying, note

a'. **The attitude of the body, and its position with regard to surrounding objects,** *e.g.*, note whether the body is lying on the ground or floor; or is lying on a bed, couch, or other article of furniture; or is seated, or supported in a semi-erect, or erect position, and, if so, how supported; or is suspended partly or completely by a ligature round the neck, &c., &c. Note the attitude of the limbs, and the position of the body in regard to surrounding objects, *e.g.*, whether the body is lying at the foot of a precipice, tree, or other high object from which it may have fallen; or is immersed wholly or partly in water; or is lying in a room, and, if so, in what part of the room, &c., &c.

b'. **The nature, condition, and position of objects in contact with or lying near the body,** *e.g.*, note if any matters are lying in, or are grasped by the hands, and, if so, their nature and condition. Note the position, nature, and condition of any ligature on the body, and the exact situation of the knot. Note whether or no any stains of blood, vomit, &c., are present near the body, and whether any weapon, or bottle or other vessel likely to have contained poison, is lying near it, preserving such

matters for further examination. Note whether any signs exist in the neighbourhood of the body indicative of a struggle having taken place, or of the employment of weapons, or generally of the presence of persons other than the deceased, at the spot about the time of infliction of the injury.

Obviously an examination of the spot where a dead body has been found, and of the position of the body in regard to surrounding objects, often affords valuable information as to the circumstances under which death occurred (see *Cases* A 11 to 14). It must be recollected, however, that the spot where the body has been found **may not** be the place at which the act was done which caused death. In such a case the question will arise: What power of locomotion remained to the deceased after the act was done which caused his death, and was this sufficient to enable him to move from the spot at which the act was done, to that where the body was found?[1] The answer to this question may, it is evident, have an important bearing on the question: Was death due to homicide, suicide, or accident?

II. External examination of the body, including examination of its coverings.

This includes—

1. **Examination of the clothes** or other coverings of the body. First, the general condition of these should be noted, *e.g.*, whether showing marks of fire, or corrosion; or wet, or stained with blood, dirt, secretions, or excretions. The clothes, &c., should then be more particularly examined; note should be taken of any cuts or rents upon them, and of the correspondence, or otherwise, of these with wounds on the body. Any peculiarities of the clothes, or of the ornaments found on the body, likely to aid in establishing its identity, should also be recorded.

2. **External examination of the body.**—It may first be pointed out that in India dead bodies are often submitted for examination in an advanced state of decomposition. Such bodies should nevertheless be examined externally, and as far as possible internally also, as even in the case of highly putrid corpses, distinct evidence as to how death was caused may often be obtained, as, for example, when this has been the result of wounds, fractures, or other violence, or of the administration of certain poisons. External examination of the body should include examination for—

a'. **Signs indicating the period which has elapsed since death took place,** *e.g.*, whether the body is warm or cold, its condition as regards rigidity, and the extent to which putrefaction has advanced. It is possible that an estimate formed as to the period which has elapsed since death may bear greatly on

[1] See Wounds, p. 53.

the questions of (1) the identity of the body, and (2) the guilt or innocence of accused persons (see *ante*, p. 18, and *Case* A 2).

b'. **Marks likely to aid in establishing the identity of the body.**—Note the probable age, state of the teeth and jaws, sex, general condition and height, the colour of the eyes and hair, and whether any, and if so what, portions of the body have been denuded of hair. Note the presence of any deformities, tumours, old scars, tattoo, sun, or other marks on the surface of the body, and of perforations for the insertion of ornaments (see *Identity*, p. 19 *et seq.*).

c'. **Marks of violence, or other external marks indicative of the cause of death**—Note if there are any stains of blood, or other matters, on the surface of the body. In the case of blood stains it is specially important to note their situation and form, as this may show that they have been produced by some individual other than deceased, *e.g*, the impress of a right hand on the right hand or forearm of the body. Note whether or no cutis anserina [1] is present, and whether any matters are sticking under the nails. Note whether the features are pale and natural, or livid and swollen, the condition of the eyes, and position of the tongue, and whether or no this shows marks of injury from the teeth. Examine the orifices of the body for presence of foreign bodies, marks of concealed punctures, and marks of corrosion, and note the nature of any fluid oozing from them. Examine, at first without dissection, the whole body for wounds or other marks of injury, not omitting to examine by palpation the bones for fractures, and to search for marks of concealed punctures under the breasts, scrotum, and eyelids, and in the armpits and nape of the neck, and, in the case of infants, in the fontanelles, and along the whole course of the spine. Then, employing such dissection as may be requisite, note in regard to all wounds or other marks of injury (1) Their exact situation. This should be recorded with reference to some fixed point on the body, *e.g.*, distance of a wound in inches from the top of the pubis or sternum, angle of the jaw, &c.; position, with reference to the hyoid bone or laryngeal cartilages of a ligature mark on the neck, &c. (2) Their exact dimensions, *e.g.*, measured in inches, the length, breadth, and depth of wounds, breadth of ligature marks, &c. (3) Their direction, *e.g.*, in a punctured wound, whether it is directed from above down or below up, and whether from right to left or left to right; and in incised or other linear wounds or marks of injury, whether or no one end is higher than the other, and, if so, whether the upper end is anterior or posterior to, or to the right or left of, the lower. In the case of certain fractures, *e.g.*, of the ribs, note whether the broken ends of the bone have been driven inwards or outwards, by the violence used to cause the fracture. (4) Their appearance, and how far this indicates (*a*)

[1] Goose skin, see Drowning, p. 78.

the method, and (*b*) the time, of their production. Under (*a*) note in the case of wounds whether the edges are contused or lacerated, or apparently clean cut, and in the latter case examine them with the aid of a lens for signs of tearing, or appearance of inversion, indicative of production by a blunt weapon. Note the general shape of any wound, contusion, or burn; this may indicate the shape of the weapon or heated object which has been used. In the case of an incised wound, compare the appearance of the two ends of the wound, with the view of ascertaining the direction in which the cutting instrument was drawn in producing it. In gunshot wounds, if two orifices exist, compare their appearance, noting any characters indicating one or other to be the orifice of entry or of exit. Note also whether or no any blackening or marks of gunpowder exist round the wound. Examine any wound for the presence of foreign bodies, preserving such as may be found. Under (*b*) note, in the case of wounds, whether or no the edges are retracted or everted, and whether or no blood or blood clots are present in the wound, or signs of inflammation exist around it. In the case of apparent contusions, note if the skin over them is abraded; examine the edge of the contused surface for changes of colour, and, by dissection, ascertain if the underlying tissues contain extravasated blood. In the case of ligature marks also, the condition of the tissues underlying the mark should be ascertained. If the injury is a burn, note the presence or absence of vesication about it, or of a line of redness, or signs of inflammation around it. Bear in mind, while conducting the examination, the characters which distinguish ante-mortem from post-mortem wounds [1] and burns,[2] and those which distinguish ecchymosis from cadaveric lividity.[3] Recollect, also, that under certain circumstances putrefactive changes may simulate signs of strangulation. There may, for example, be protrusion of the tongue, due to such changes; and putrefactive swelling against a string loosely tied round the neck, may result in the production of a depressed mark, similar to that left by the ligature in death from strangulation.

In the case of infants the external examination should, in addition, include examination for

(*d'*) **Signs indicating degree of maturity,** viz., length and weight, condition of the eyes, condition of the skin, nails, and scalp hair, and position of the middle point of the body (see *Inheritance, Question VI.*, p. 346), and

(*e'*) **Signs indicating live birth, or the reverse,** *e.g.*, exfoliation of the scarf skin, condition of the umbilical cord, presence of signs of intra-uterine maceration (see *Infanticide, Question I. c*, and *d*, p. 360).

[1] See p. 47 *et seq.* [2] See p. 94. [3] See p. 41.

III. Internal Examination of the Body.

For details as to the procedure, &c., advisable in conducting this, see Appendix XXIX. Here the following general questions in regard to it may be considered, viz. 1. The order in which it should be conducted. 2. The necessity for its being complete. 3. The inference to be drawn from its results; and 4. The special points to be attended to in examining the bodies of infants.

1. **As to the order in which the examination should be conducted**—(*a*) If on the surface of the body marks of violence exist of such a nature as to indicate injury to the organs contained in any particular cavity, that cavity should be first dissected, and the exact effect of the violence ascertained. Incisions made through the skin for this purpose should avoid wounds likely to have penetrated the cavities underlying them, so that there may be no interference with the subsequent tracing of the exact course of the wound. (*b*) In other cases, where a particular cause of death is suspected, that cavity should be first dissected in which appearances indicative of the cause of death are most likely to be found, *e.g.*, in cases where it is suspected that death has been due to asphyxia, the thorax, and in cases of poisoning, the abdomen, should be the cavity first dissected. (*c*) In cases where the cause of death is doubtful, the three chief cavities of the body should be dissected in the following order: 1. The head; 2. The thorax; and 3. The abdomen. (*d*) In all cases before opening and dissecting the thorax, the cavity of the abdomen is to be opened, and (without further dissection) the position of the diaphragm, and general appearance of the contents of the abdomen ascertained. Should this preliminary inspection indicate that the cause of death is connected with the abdominal viscera, dissection of the abdomen should be proceeded with; if not, it should be reserved until after the thorax has been dissected. (*e*) It should specially be recollected that the condition as to fulness or the reverse of the cavities of the heart at the time of death, can only be with certainty ascertained, when these are opened before removing this, or any other organ from the body.

2. **The examination should be complete.**—Even if the condition of the parts first examined appears sufficiently to account for death, the remainder of the body should be examined, so that, in fact, you may be in a position to say, not only what was, but also, as far as possible, what was not, the cause of death. Cases are on record, in which a complete post-mortem examination of a body has disclosed indications of a cause of death other than that indicated by the condition of the parts first examined. The appearances first noted may, for example, indicate that death was due to mechanical violence, or to drowning, and yet on

further examination, indications may be found of death from poison (see *Cases* A 18) Or, again, the appearances first noted may indicate death from hanging, and yet further examination may show that death was due to strangulation (*Cases* A 23), or to mechanical violence (*Cases* A 24), and that the appearances indicative of death from hanging have been fabricated in order to make the case seem one of suicide. Again, suppose the post-mortem appearances show clearly that death was due to a particular lesion, your examination is not complete unless you have noted all particulars which may assist in arriving at an opinion as to whether the fatal lesion was the result of disease, or of violence, and if the latter, whether the violence was accidental, suicidal, or homicidal.

3. **No opinion should be given as to the cause of death that is not fully warranted by the appearances observed.**—It is quite possible that, on post-mortem examination, you may fail to discover appearances sufficient to account for death. In such a case, a negative opinion only should be given. You are not justified in reporting death to be due to "natural causes," simply because you have failed to find post-mortem appearances indicative of death from a non-natural cause. Again, on examination, you may find that death was due to a lesion, *e.g.*, an effusion of blood into the substance of the brain, which may either have been the result of violence, or the result of disease; and the post-mortem appearances may do no more than indicate that the fatal lesion was more probably the result of one than of the other. In such a case, your opinion should be a guarded one, and be accompanied by the reasons which lead you to consider it to be more probable that disease or violence, as the case may be, was the cause of the lesion.

4. **In examining the bodies of infants,** it must be borne in mind that the examination must not only be directed towards ascertaining (1) the cause of death, but also towards ascertaining (2) the degree of maturity of the infant, and (3) whether or no it survived its birth. The points bearing on the first of these two additional questions will be found under *Inheritance* (*Question VI.*, see p. 346), and those bearing on the second under *Infanticide* (*Question I.*, see p. 353).

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CHAPTER III.

WOUNDS AND MECHANICAL INJURIES.

THE principal medico-legal questions connected with these are—

I. WAS A WEAPON USED, AND IF SO, WHAT WEAPON, OR WHAT KIND OF WEAPON? II. WAS THE INJURY INFLICTED DURING LIFE, OR AFTER DEATH? III. WAS THE INJURY THE RESULT OF ACCIDENT, OR SELF-INFLICTED, OR INFLICTED BY ANOTHER? IV. WHAT RESULTS FOLLOWED, OR ARE LIKELY TO FOLLOW, FROM THE INJURY?

QUESTION I.—WAS A WEAPON USED, AND IF SO, WHAT WEAPON, OR WHAT KIND OF WEAPON?

A conclusion arrived at in regard to this question may be of importance, because—

1. By sects. 324 and 326 of the Indian Penal Code, the causing of hurt, or grievous hurt, by certain specified means, is made an offence punishable more severely than when such means have not been used. Among the means specified in these sections, are "Any instrument for shooting, stabbing, or cutting, or any instrument which, used as a weapon of offence, is likely to cause death"

2. It may affect the question of the guilt or innocence of an accused person For example, the evidence may show that the accused, if guilty, used a weapon produced; but the characters of the injury may show that it could not have been caused thereby (see Presence or Absence of Foreign Bodies in Wounds, *post*, p. 46).

The chief points which must be taken into consideration in arriving at a conclusion on Question I, are—1. The class to which the wound or injury belongs. 2. The presence or absence of foreign bodies in the wound, and 3. The condition of the weapon alleged to have been used.

Question I.—1. The class to which the wound or injury belongs.

For medico-legal purposes, wounds and mechanical injuries may be divided into two classes, viz. those which are not, and those which are, accompanied by visible solution of continuity. The latter class may again be divided into, incised, lacerated and contused, punctured, and gunshot wounds.

1. **Injuries unaccompanied by any visible solution of continuity** may be accompanied by serious internal solutions of continuity, *e.g*, fractures of bones, or rupture of some important organ, such as the spleen, and hence may be of any degree of severity, from extremely slight, to mortal wounds. The medical term a wound, includes all solutions of continuity, whether external or internal. Courts of law in England have, however, held that the legal definition of the term, a wound, is an external injury in which the true skin is divided. Hence injuries of the class now under consideration, no matter how severe, are not wounds in law.

Injuries without visible solution of continuity are usually, but not invariably, accompanied by **ecchymosis**, *i.e.*, discoloration of the skin from effusion of blood into the subcutaneous cellular tissue. **Ecchymosis** shows itself as a dark dull reddish-blue discoloration of the skin, which in about twenty-four hours begins to change colour, becoming lighter, and changing in tint to violet, then to green, and lastly to yellow, and finally disappears altogether in about five or six days. These changes in colour commence at the circumference of the patch, are supposed to be due to dilution of the effused blood by the serum of the cellular tissue and subsequent absorption, and occur only during life. Ecchymosis may, to a certain extent, be simulated by cadaveric lividity, a discoloration of the skin due to post-mortem changes, especially when this, owing to the pressure of a sheet or other covering on the body, occurs in stripes resembling marks of flogging. **Cadaveric lividity**, however, (*a*) affects dependent parts; (*b*) is usually of great extent; and (*c*) is unaccompanied by extravasation of blood. An incision through the skin and examination of the underlying cellular tissue, therefore, will always disclose the true nature of the discoloration Attempts are sometimes made to **simulate ecchymosis** by applying marking nut juice, or some other irritant, to the skin; such applications, however, usually produce blistering, or a papular eruption, easily distinguished from ecchymosis. **Ecchymosis** (*a*) may occur at a distance from the spot to which violence has been applied, *e.g*, at the seat of fracture of a bone broken by indirect violence; (*b*) may occur in spots (petechiæ), without violence, as in some diseases, *e.g.*, scurvy, and some cases of snake poisoning; (*c*) other things being equal, is in amount less, the better the

bodily condition of the individual injured, and greater the looser the texture of the skin at the seat of injury; lastly, (*d*) its disappearance during life is apt to be extremely slow in old persons; is more rapid the better the bodily condition of the sufferer; and may be hastened by external applications (see *Case* A 19). After death it may disappear from, or be masked by decomposition, or from the effect of antiseptic agents applied to the body.

Serious, even mortal, injuries may be caused by mechanical violence without ecchymosis being produced, especially when the violence has been applied to a yielding part, *e.g.*, the anterior abdominal wall. In the absence of ecchymosis, the fact of the existence of an internal injury caused by external violence, is, during life, a matter of surgical diagnosis. After death, the existence of an internal solution of continuity may be ascertained by dissection, and, in the absence of ecchymosis, its connection with external violence is sometimes indicated by bruising of the parts lying between it and the surface. If such bruising is absent, as well as ecchymosis, the question whether or no the solution of continuity discovered has been caused by external violence, may be a difficult one to decide, requiring consideration of points such as (*a*) The freedom, or otherwise, from disease of the affected part; (*b*) The situation of the affected part, and its degree of liability to rupture from causes other than external violence;[1] and (*c*) The history of the case.

Injuries unaccompanied by visible solution of continuity may of course be produced without a weapon. If a weapon has been used, it will probably have been a blunt or rounded one, such as a stick or club. Frequently the shape, &c., of the weapon or instrument employed can be inferred from the shape and situation of the patch or patches of ecchymosis. Weapons commonly employed in India in the production of severe injuries of this class are (1) A bamboo staff or club, often shod with iron, *láthi*, or *sonta*, or when bound with iron, *lohabundee*. Harvey[2] mentions that about 32 per cent. of the medico-legal cases reported in Bengal, &c., during the three years ending 1872 were *láthi* wounds; and (2) The rice-pounder, a club usually of hard wood, about 3½ feet long, and 1½ to 2 inches in diameter, shod at one end with a thin iron plate about 1 to 1½ inch long. This, Gribble[3] states, is a common weapon of assault in the Madras Presidency. Instruments more or less frequently used in India in producing slight injuries of this class, requiring special mention are (1) Shoes—beating with a shoe is supposed to add insult to the injury; and (2) Ropes or cords, used either for the purpose of tying up the sufferer as a mode of

[1] See Injuries to the brain, thorax, abdomen, &c., *post*, p. 59 *et seq.*
[2] Harvey, 'Bengal Med. Leg. Rep.' 1870–72, p. 20.
[3] Gribble's 'Medical Jurisprudence,' p. 28.

torture, or to secure the victim during the infliction of other injuries. Usually the arms are secured behind the back by binding together the elbows or wrists.

2. **Incised wounds.**—In examining an apparently incised wound with the object of ascertaining the kind of weapon, if any, used in producing it, it is important to note (1) the situation of the wound, (2) the appearance of its edges, and (3) its length and depth in different parts. (1) **Situation.** An apparently incised wound situated on a part where the skin closely overlies a bone, or sharp ridge of bone, may be produced without a weapon or by a blunt weapon. Blows with the fist, for example, over sharp ridges of bone such as the chin, or orbital ridge, or blows with a club on the scalp, may produce wounds closely resembling incised wounds. Wounds caused in this way are generally, but not invariably, vertical to the bone. (2) **Appearance of edges.** These should be examined with a lens. Sharp, clean cut, uninverted edges, indicate the use of a sharp-edged weapon; tearing and inversion indicate the employment of a blunt weapon, or production without a weapon. (3) **Length and depth in different parts.** Long incised wounds indicate the use of a sharp-edged weapon, and may either be caused by a single blow from one with a long, tolerably straight edge, such as a sword, or by a drawing cut from one with a short edge, such as a razor. In the former case, the method of production is often indicated by the underlying bones being clean cut through; and in the latter by the wound tailing off at one end into a superficial scratch (see also in regard to this, *Question III*, 2, *direction of the wound*). Weapons of the axe or hatchet class usually produce comparatively short incised wounds; either deep, or accompanied by indentation and extensive fractures of the bones beneath. Cutting instruments with a concave edge and projecting point often cause linear wounds resembling a punctured wound at one end, and gradually decreasing in depth towards the other end. Or if the wound has been inflicted on a curved surface, the puncture caused by the point, and the incised wound caused by the edge, may be separated by an unwounded portion of the skin. Weapons of assault more or less commonly used in India, sharp-edged, and producing incised wounds are (1) Light, short-edged weapons, such as the razor, *ustara*, and the knife, *churi*. (2) Heavy, short-edged weapons of the hatchet class. These include the axe, *kulhári*, and the *garási*, *gandása*, or *tarash*, an axe-like weapon with a long handle, something like a halberd. Weapons allied to this class are the *phaora* or *kora*, a species of spade with the blade set at a considerable angle to the handle, and the Goorkha *kukri*, a short, heavy, convex-edged sword. McLeod also mentions as belonging to the spade class the *khurpa*, or grass-cutter's knife. (3) Long-edged weapons, represented by the curved sword *talwar*,

or the straight sword *kirich;* and (4) Weapons with a concave edge and projecting point, such as the billhook, *dao, koita,* or *aut,* and the sickle, *hussolee, dáránti,* or *ela.*

3. **Contused and lacerated wounds.**—These are often the result of injury by means other than the employment of a weapon. Thus they may result from (1) Injuries by broken glass—broken-glass wounds, however, if slight, are apt to resemble incised wounds. (2) Falls on some projecting, more or less sharp object. (3) Injuries from wild animals; or (4) Machinery and railway accidents. Severe contused and lacerated wounds are often accompanied by very little hæmorrhage. Slight non-accidental lacerated wounds, produced without a weapon, may be the result of injuries inflicted in forcibly tearing out ornaments, or by the teeth or nails. If a wound of this class has been produced by a weapon, and much contusion or laceration is present, the indication of course is that a rounded or blunt-edged weapon has been used. Sharp-edged knives, it should be noted, if used with considerable force, cause bruising and laceration of the parts divided.[1]

4. **Punctured wounds.**—Wounds more or less resembling punctured wounds may, especially if the edges of the puncture are lacerated or bruised, be caused without a weapon, *e.g.,* by accidental injury from projecting nails, or from fragments of crockery. If the edges of the puncture are free from laceration or contusion, the indication is that a sharp-pointed weapon has been employed. Sometimes, but not always, the shape of the weapon which has been used is indicated by the shape of the puncture in the skin. Dupuytren found, however, that cylindrical pins produce elongated openings.[2] The obliquity or directness of the thrust, and also the state of tension or relaxation of the skin, may affect the shape of the puncture, and hence two punctures from the same weapon may differ in shape. Very often also, owing to the elasticity of the skin, a punctured wound is of less diameter than the weapon which has been used. Sometimes in a punctured wound, the broken-off point of the weapon employed is found. Punctured wounds are occasionally found in concealed situations, *e.g.,* in the rectum or vagina, in the armpit, or under the upper eyelid. A minute puncture in certain situations, *e.g.,* over the fontanelles in infants, or in the nape of the neck, may indicate a mortal wound. The existence of several punctured wounds of course very strongly indicates the employment of a weapon, and if all are similar in size and shape the probabilities are in favour of their being due to repeated thrusts with the same weapon. Weapons more or less commonly employed in India giving rise to punctured wounds are, besides knives and weapons of the billhook class already mentioned, (1) daggers, *katari,* of various shapes—in some of these the

[1] Ogston's 'Lectures,' p. 420. [2] *Ibid.,* p. 416.

handle is transverse to the axis of the blade; (2) the spear, *bhala*, *barchi*, or *sulfi*; and (3) arrows. Arrow wounds, it may be pointed out, are frequently fatal. Harvey records in Bengal, &c., in the three years ending 1872, fifteen fatal cases out of a total of twenty-five. *Case* A 17 illustrates the great penetrative power frequently imparted to these projectiles. The pickaxe, *kudáli*, may also cause a punctured wound, probably with much contusion; and punctured wounds may be produced by thrusts with a pointed bamboo (see *Case* A 24 *e*).

The same weapon may cause wounds belonging to two or more of the above four classes.—A heavy weapon of the billhook class may, for example, produce all four varieties. Hence the existence on the body of the same individual of wounds belonging to two or more of these four classes, does not necessarily indicate that two or more weapons were employed, or that more than one person was concerned in their infliction.

Injuries inflicted by animals may produce wounds belonging to, or resembling, wounds of any of the above four classes. Injuries without visible solution of continuity, often severe enough to cause death, are sometimes caused by elephants kicking, trampling on, or butting, the injured individual, or by the animal seizing the individual with his trunk and dashing him forcibly on the ground. Apparently incised wounds may be caused by the tusks of the wild boar. Harvey describes such wounds as "long clean rips," and mentions a case in which a wound so produced, on the inside of the left thigh, was twelve inches long, three deep, and one broad, and, dividing the femoral artery, caused death by hæmorrhage. Fatal contused and lacerated wounds may be caused by various animals; thus they may result from a bite, or from a blow with the paw, or from injury by the claws, of an animal of the tiger class, in which case the neck is often the seat of injury; or they may be the result of a bite from a crocodile, or of an injury inflicted by a bear, in which last case the scalp is often found greatly torn. Again, contused and lacerated wounds may be the result of injuries inflicted by domestic animals, *e.g.*, a kick from a horse or cow. Punctured, or punctured and lacerated wounds, may be caused by the tusks, or more frequently by the horns, of animals. Harvey states that in Bengal, &c., in three years, twenty cases of gores by horned cattle, ten of them fatal, were reported. On the whole, injuries caused by animals are so characteristic in appearance that there is seldom any difficulty in deciding as to their origin.

5. **Gunshot wounds** resemble contused and lacerated wounds in character, and indicate, of course, the employment of a firearm, but not necessarily the discharge therefrom of a hard projectile. If the wound is **single**, it may have been caused by a firearm loaded with powder and wadding only, if the weapon has been discharged near the body. Nearness of the weapon to the body

at the time of discharge is indicated by blackening of the skin from the gunpowder, or by charring or blackening of the clothes at the seat of injury. A single wound, however, may be caused by a firearm loaded with a hard projectile, which in such a case will often, but not invariably, be found lodged in the wound. **Two orifices** caused by the same discharge, indicate the employment of a hard projectile. When two orifices are present, the orifice of entry will often, but not always, be found to be smaller than that of exit. Depression and absence of raggedness at the edges indicates the orifice to be one of entry; raggedness and eversion, one of exit. More than two orifices may be caused by one projectile, *e.g.*, when this has entered the body after traversing a limb, or has split up against a sharp ridge of bone into two pieces, each finding a separate exit. Harvey mentions a case in which a wound in the neck, produced by a thrust with a "pointed perfectly circular bamboo," was mistaken for a gunshot wound (see *Case* A 24 *e*).

Question I.—2. The presence or absence of foreign bodies in the wound.

Foreign bodies present in a wound may be either such as do not specially indicate the employment of a weapon, *e.g.*, particles of dirt or fragments of glass; or bodies which show that a weapon has been used, *e.g.*, the broken-off point of a knife or dagger, or the projectile or wadding of a firearm. Deep punctured and gunshot wounds should specially be searched for foreign bodies of this latter description, which, if found, should be preserved for production in Court, as they are frequently of great importance as links in a chain of evidence. For example, the broken-off point of a knife found in a wound, may exactly fit a knife alleged to have been used, or this may have a perfect point, showing that it was not the weapon employed. Again, a projectile found in a gunshot wound may fit, or be too large to have been discharged from, the firearm alleged to have been used; or a piece of paper or cloth, used as wadding, and found in a gunshot wound, may correspond to similar fragments found in possession of the accused. Foreign bodies are not always found in gunshot wounds, a projectile, for example, may have completely traversed the body; or may have lodged and subsequently dropped out, as sometimes happens when the wound is shallow, or when a portion of clothing has been carried into the wound with the projectile.

Question I.—3. The condition of the weapon alleged to have been used.

This, if a firearm, may show signs of recent discharge. Recently discharged firearms will be found blackened inside the barrel, from the residue left by the gunpowder after ignition. This residue consists mainly of finely divided carbon and potassium sulphide, and yields to water a dark-coloured liquid, alkaline in reaction, and which, after filtration, strikes a black colour with a solution of a lead salt. After a time the potassium sulphide becomes oxidised into potassium sulphate, rust (hydrated ferric oxide) also forming. Hence, owing to the oxidation of the sulphide into a sulphate, washings from a firearm which has been some time discharged, may show no alkaline reaction, and give no blackening with lead salts. Again, the weapon alleged to have been used may show signs of recent fracture, or be bent or otherwise injured, as the result of its use; or the fact of its use may be indicated by the presence on it of stains of blood, or of adhering fragments of hair or other fibres (see *post*, p. 70, *et seq.*)

QUESTION II.—WAS THE INJURY INFLICTED DURING LIFE OR AFTER DEATH?

Injuries inflicted after death although often the result of accident, may also be the result of design, the motive being either concealment of a crime, or fabrication of evidence in support of a false charge. Further, the fact that an injury severe enough to cause death was inflicted during life, is evidence in favour of its having been the cause of death. Hence the importance of this question. Ante-mortem injuries are distinguished from post-mortem injuries by the presence of signs indicative of vital action. These may conveniently be considered under the heads of (1) Ecchymosis; (2) Effusion of blood; and (3) Other signs.

1. Ecchymosis.

Blows inflicted during life, if severe, are generally, but not invariably, followed by ecchymosis. Ecchymosis may appear even if the individual has lived only a very short time after receipt of the injury; and further, ecchymosis from blows inflicted during life, may not appear until after death has taken place. The presence of ecchymosis, however, does not necessarily indicate that the injury producing it was inflicted during life. Christison found that blows inflicted on dead bodies, within two or three hours after death, were followed by ecchymosis, not distinguishable from ecchymosis the result of blows inflicted during life. If the individual has lived for some time, say more than twenty-four hours after receiving the injury, changes in

colour will probably be found at the circumference of the ecchymosed patch (see *ante*, p. 41), and the injured part will probably be found swollen. The presence at the circumference of the ecchymosed patch of changes in colour of the kind described at p. 41, and the presence of swelling of the injured part, show that the injury was inflicted some time before death.

2. Effusion of Blood.

In a dead body the blood remains fluid for some time after death, rarely beginning to coagulate until four hours, and sometimes not until twelve hours, after death. Hence an injury inflicted after death, while the blood is still fluid, may be followed by effusion of blood. Owing, however, to arrest of the heart's action, no arterial spouting occurs, and the quantity of blood effused is much less than would be effused from a similar injury inflicted during life. Further, blood effused from a wound, made more than ten minutes after death, rarely coagulates. Hence, marks of arterial spouting indicate infliction during life. Much hæmorrhage also indicates ante-mortem infliction, and if the blood effused is found coagulated, the presumption is strong that the injury was inflicted either during life, or very shortly after death. Post-mortem infliction is indicated if the effused blood is found fluid, but not necessarily by the quantity of effused blood being small, seeing that severe contused and lacerated wounds, inflicted during life are sometimes followed by but little hæmorrhage.

3. Other Signs.

Retraction and eversion of the edges of the wound follows the infliction of an incised wound made during life or shortly after death. Wounds other than incised wounds, inflicted during life, exhibit this character in proportion to the closeness with which they approximate in nature to incised wounds. Hence in incised wounds, or wounds approaching in character to incised wounds, indications of infliction during life, or shortly after death, are:—(*a*) retraction and eversion of the edges of the wound; (*b*) hæmorrhage into the wound, and into the cellular tissue around it; and (*c*) the presence of coagula.

Signs of inflammatory or reparative action.—The existence of these about a wound of course proves ante-mortem infliction, and the more developed these signs are, the longer probably the period which intervened between the infliction of the wound and death. It should, however, be recollected that the rapidity with which inflammatory action sets in, and its amount, are likely to be greater, the more the amount of contusion and laceration about the wound.

The following table summarizes the inferences as to ante- or post-mortem infliction, derivable from the character of wounds:—

CHARACTER	INDICATION.
Presence of	**That the wound was inflicted**
Signs of inflammation round the injury. Changes of colour at the circumference of a patch of ecchymosis.	Certainly before, and probably more than twenty-four hours before, death.
Marks of arterial spouting. Extensive hæmorrhage.	Before death.
Coagula.	During life, or very shortly after death.
Retraction and eversion of the edges of the wound. Ecchymosis.	During life, or probably not more than three hours after death.
Complete absence of all the above characters.	Probably more than twelve hours after death.

QUESTION III.—WAS THE INJURY THE RESULT OF ACCIDENT, SELF-INFLICTED, OR INFLICTED BY ANOTHER?

The importance of this question is obvious. In considering it, it is desirable to bear in mind that in India severe, even mortal, injuries are sometimes inflicted on an individual with his consent, by another or others, for the purpose of supporting a false charge (see *Cases* A 8, 9, and 10). The chief points bearing on Question III. are:—

1. The nature and situation of the injury.
2. The direction of the wound.
3. The number of wounds or injuries.
4. The position and surroundings of the injured individual.

Question III. 1. The nature and situation of the injury.

In many cases, these characters afford no indication as to how, or by whom, the injury was inflicted. A presumption more or less strong, however, arises.

Against self-infliction, in the case of stabs passing right through the body, and cut throat extending to the vertebræ, these being rarely self-inflicted wounds. So also stabs, and incised wounds on the back, and gunshot wounds, unaccompanied by any blackening of the skin or scorching of the clothes, are

only likely to be self-inflicted wounds, if some special contrivance has been used to fix, or in the case of a gunshot wound to fix and discharge from a distance, the weapon employed. Contused wounds, again, especially if severe, are only likely to have been self-inflicted if the individual is insane, or the case is one of suicide by precipitation from a height.

In favour of self-infliction.—A strong presumption in favour of this arises in the case of a gunshot wound caused by the discharge of a firearm in the mouth. Again, in the case of incised wounds, if these are all slight the presumption is, to a certain extent, in favour of self-infliction (see *Cases* A 7); and it may be further noted, that severe self-inflicted incised wounds, commonly tail off greatly at one end, into a mere superficial scratch.

In favour of infliction by another, in cases where severe incised wounds are accompanied by cuts on the hands of the injured individual, and in female subjects—if the circumstances exclude accident—in cases of wounds of the genitals, or mutilating wounds of the nose, ears, or breasts. In India, wounds in females in the situations just mentioned, indicate jealousy, or punishment for adultery as the motive for their infliction; and wounds of the genitals in male subjects often also indicate the existence of the last-mentioned motive.

Question III. 2. The direction of the wound.

It may first be pointed out that while a non-self-inflicted wound may have any direction, a self-inflicted wound usually has a particular direction, dependent on the part wounded, and the hand employed. Hence it is important, where possible, to ascertain whether the injured individual is or was right or left-handed, or ambi-dextrous. Next, in order to obtain the fullest information from the direction of the wound, an endeavour should be made to determine its beginning and ending; this, of course, presents no difficulty in the case of punctured wounds, and non-traversing gunshot wounds. In traversing gunshot wounds, the beginning and ending of the wound are indicated by respectively the orifice of entry and the orifice of exit (see *ante*, p. 46). It may here, however, be remarked that it by no means follows that the direction of such a wound is represented by a straight line drawn from one orifice to the other. For example, a projectile may be deflected by a bone or by tough fascia, and take a circuitous course, *e.g.* may be deflected by a rib, and pass half round the body without penetrating the chest, or pass half round the head without penetrating the skull. In the case of incised wounds made by a drawing-cut, if one end is abrupt, deep, and unbifurcated, and the other shallow, and tailing off, or bifurcated, the probabilities are that the former is the beginning,

and the latter the ending of the wound. **Self-inflicted incised wounds**, as a rule, (1) end on the same side as the hand employed, and (2) begin from below if on the lower part, or from above if on the upper part of the body. **Self-inflicted incised wounds of the throat**, as a rule, possess the first of these characters, but may or may not possess the second, *i.e.* they may be transverse, or run from above down,[1] or from below up. Self-inflicted stabs and gunshot wounds commence, as a rule, on the same side as the hand employed. **Homicidal wounds** may have any direction, and are specially liable to have the same direction as self-inflicted wounds, if the assailant was standing behind his victim, at the time of inflicting the injury. Frequently in homicidal wounds, the direction of the wound indicates the relative position of the assailant and victim; it must be recollected, however, that the direction of the cutting edge of some weapons is transverse to the line of the handle. This is the case in the carpenter's adze, and the mattock or spade, *phaora* or *kora*, commonly employed by cultivators in India.

Question III. 3. The number of wounds or injuries.

A single wound or injury may be the result of accident, self-inflicted, or inflicted by another. When many wounds are present, self-infliction and accident are, to a certain extent, contra-indicated. **Numerous wounds** may, however, be

1. **Self-inflicted. Several incised wounds**, all slight, are sometimes self-inflicted, with the object of averting suspicion (see *Case* A 7 *a*); or of supporting a false charge (see *Case* A 7 *b*). In suicidal cases also, *e.g.* cut-throat, one severe incised wound is sometimes found accompanied by other slight cuts. More than one severe incised wound may, of course, be self-inflicted, but the greater the number, the stronger the indication in favour of homicide. **Several contused wounds** are only likely to have been self-inflicted in the case of insanes, or in cases of suicide by precipitation from a height. Suicide by precipitation excluded, **self-infliction is contra-indicated**, if each of two or more wounds is of such nature as to cause immediate insensibility, or immediate death. Very severe wounds, however, may not cause immediate death, or immediate insensibility, and hence the existence of two such wounds may still be consistent with self-infliction; *e.g.* Hayes Agnew concludes from recorded cases that it is possible for a suicide to shoot himself "first in the

[1] K. McLeod, from the cases reported in Bengal in 1869, considers that suicidal wounds of the throat are generally high up on the neck, between the hyoid and thyroid, more on the right than on the left side, and are either transverse or incline from below upwards.—Beng. Med. Leg. Rep. 1869, p. 57. Casper, however, while admitting the difficulty of ascertaining the commencement and end of wounds, states: "In suicidal wounds of the throat the wound certainly usually runs from left to right and from above downwards."—ii. p. 13.

head, and within the lapse of a minute inflict a similar wound on the heart," or *vice versâ*.[1] Very great caution, therefore, must be exercised in drawing a positive inference against self-infliction, simply from the fact that more than one severe wound is present on the body.

2. The result of accident, *e.g.* a fall from a height, an accident from machinery, &c. In such a case, however, homicidal violence is not contra-indicated, unless all the injuries are to be accounted for by the supposed accident. Thus when the accident indicated is a fall from a height, and there are no projecting objects against which the body could have struck during its descent, severe injuries on both sides of the head contra-indicate accident (see *Case* A 15).

Question III. 4. The position and surroundings of the injured individual.

Under this head should be noted—

1. The position and attitude of the body and its relation to surrounding fixed or large objects.—This may directly indicate self-infliction or accident, as, for example, when the body is found at the foot of some high object, from the top of which it may have fallen. It must be borne in mind, however, that an attempt is sometimes made to conceal murder, by placing the body of the victim in such a position as to point to accident or self-infliction as the cause of the injuries. With this object the body (as in *Case* A 15) may be placed at the foot of a high tree, or on a railway line, &c. In such cases discrepancies between the nature and situation of the injuries, and the method of production indicated by the position of the body, point indirectly to the employment of homicidal violence. In other cases also indirect indications of the employment of homicidal violence may be afforded by the position and attitude of the body. This is so when the injuries present are of such a nature as to make it improbable that the position and attitude in which the body was found resulted from effort on the part of the injured individual. For example, the body may be found at a distance from the place of infliction of the injury, or in an attitude widely differing from that in which it must have been in, directly after its receipt, *e.g.* the cause of death being fracture of the skull from a blow on the back of the head, the body is found in an upright position, supported against a high thick hedge at its back. The possibility, however, even when the injuries are extremely severe, of considerable effort on the part of the injured individual must not be overlooked. It has already been pointed out (see *Question III.* 3) that a certain amount of power of voluntary movement may still be retained after receipt of very severe wounds, and it

[1] 'Annals of Surgery,' vol. vi. p. 152.

now may be pointed out that very considerable power of locomotion may remain even after the receipt of almost immediately mortal injuries. Thus Taylor mentions a case in which a man ran eighteen yards after receiving a stab penetrating the heart, and another in which it is probable that a girl ran twenty-three yards, and climbed over a gate, after receiving a wound which divided the carotid artery and jugular vein (see also *Case* A 16, and *Injuries of Regions, post*, p. 58).

2. The condition of the surface of the body, or of the clothes or other coverings thereof.—Important points to note under this head are: (*a*) Peculiarities of the clothes likely to have modified the injury received, or to affect the condition of the weapon used, *e.g.* a thick turban may cause a severe blow from a blunt weapon to produce a simple, instead of a compound fracture of the skull; or fibres derived from an article of clothing worn over the injured part, may be found adhering to a weapon and thus indicate it to be the one which was used. (*b*) Stains of blood or other matters. It is possible that these by their nature or position may indicate homicidal violence, *e.g.* stains of seminal fluid on the clothes or body of a female corpse, or a mark of a bloody right-hand on the right-hand or arm of the injured person. Again, in the case of gunshot wounds, blackening of the skin at, or of the clothes worn over, the seat of the wound, by indicating nearness to the body of the weapon at the time of its discharge, is confirmatory evidence in favour of self-infliction; just as the absence of such marks by affording a contrary indication, is evidence of more or less weight against self-infliction. Stains of mud or dust on the surface of the body, with abrasions in certain situations on the skin, may indicate dragging of the body along the ground, and afford possibly, confirmatory evidence of homicidal violence. (*c*) Cuts or rents on the clothes. Want of correspondence in situation between cuts on the clothes, and wounds on the body, is often found when the wounds have been self-inflicted for the purpose of supporting a false charge, or averting suspicion.

3. The nature, position and condition of objects on and near the spot where the body was found or the injury inflicted.—The objects found may be weapons, sharp stones, articles of clothing, fragments of clothes, &c., and these or other objects may bear stains of blood. Again, near the body or place of injury may be found bullet-marks, footprints of persons other than the deceased, or marks indicating that a struggle has taken place. If a weapon is found its position is of importance. This may indicate self-infliction, as for example when the weapon used is found tightly grasped in the hand of the dead body (see *Case* A 14). A weapon, however, found loosely lying in the hand of a dead body, may have been placed there, with the view of fabricating evidence in favour of suicide (see *Case* A 13).

The discovery of the weapon used, at a distance from the body, indicates homicide, in proportion to the improbability of its having been placed where it was found, by the deceased. Here, obviously, the question of what power of effort or locomotion remained to the deceased after receipt of the injury, must be considered. As regards the nature and **condition of the weapon** found, it may be pointed out that peculiarities in its shape, &c., by their agreement with the shape of the wounds on the body, may indicate it to be the weapon which has been used, and this again may be confirmed by the weapon showing signs of recent use (see *Question I.* 3). Marks, again, indicating ownership, present on the weapon, may be important as evidence in favour of the guilt or innocence of the accused. The non-discovery of the weapon used, especially if the injuries are likely to have caused rapid death or insensibility, obviously points to homicide. As regards **sharp stones**, the presence or absence of these is of importance as indicating the possibility or otherwise of the injuries being caused by forcible accidental contact therewith. A compound fracture of the skull, for example, a common result of a blow with a blunt weapon, may be caused by a fall on a sharp stone, but is an exceptional result of a fall on a flat surface.[1] As regards **articles of clothing or fragments of clothes**, the presence of these near the body, or grasped in the hands of the deceased, may indicate that a struggle took place shortly before death. between the deceased and some other person, and thus indicate homicide. **Fragments of hair**, again, belonging or not belonging to the injured person, may be found in similar situations, or adhering to weapons, and may prove of much importance in evidence. **Stains of blood** may be found on a weapon indicating its recent use, or on other objects in the neighbourhood of the body or the spot where the injury was inflicted. Sometimes the appearance and position of such stains is important as indicating the circumstances under which the injury was inflicted (see *Case* A 11). **Bullet-marks or shot-holes**, by their situation, may indicate the position of the assailant at the time the weapon was discharged (see *Cases* A 12 and 13). As regards **footprints**, Ogston[2] remarks that the impression left by the naked foot varies in the same individual according as to whether he was standing, walking or running at the time.[3] Lastly, objects in the neighbourhood may be found overturned, broken, or showing marks of injury, pointing to a struggle having taken place.

[1] Harvey's 'Beng. Med. Leg. Rep.,' p. 53. [2] 'Lectures,' p. 63.

[3] Casts of footprints may be obtained by Hugolin's method, viz., heat the ground by holding over it a pan of burning charcoal, then dust stearic acid into the foot-print, let this solidify, and from the mould thus obtained take a cast in plaster of Paris.

QUESTION IV.—WHAT RESULTS FOLLOWED, OR ARE LIKELY TO FOLLOW, FROM THE INJURY.

It may first be noted that, for medico-legal purposes in India, the results liable to follow wounds and mechanical injuries may be stated to be: 1. Death; 2. "Grievous hurt;" and 3. Hurt not amounting to "grievous hurt."

1. **Death.**—If an act done by another results in death, the doer of the act may be charged with the offence of committing "culpable homicide," as defined in sect. 299 of the Indian Penal Code,[1] and may be convicted of this offence even if death followed as an indirect result of the injury[2] (see sect. 299, Explanations 1 and 2). Culpable homicide, according to the circumstances of the case, may or may not amount to murder. Again, when a person has committed suicide, any one who has abetted him in doing so is punishable under sects. 305 or 306 of the Code. By sect. 305 abetment of suicide may be punished with death, if the suicide was under eighteen, or was insane, delirious, or intoxicated at the time. Attempts to commit murder, or culpable homicide, are punishable under respectively sects. 307 and 308 of the Code, and attempts to commit suicide under sect. 309.

[1] Sect. 299 of the Indian Penal Code is as follows:—"Whoever causes death by doing an act with the intention of causing death, or with the intention of causing such bodily injury as is likely to cause death, or with the knowledge that he is likely by such act to cause death, commits the offence of culpable homicide.

"Explanation 1. A person who causes bodily injury to another who is labouring under a disorder, disease, or bodily infirmity, and thereby accelerates the death of that other, shall be deemed to have caused his death.

"Explanation 2. Where death is caused by bodily injury, the person who caused such bodily injury shall be deemed to have caused the death, although by resorting to proper remedies and skilful treatment the death might have been prevented.

"Explanation 3. The causing of the death of a child in the mother's womb is not homicide. But it may amount to culpable homicide to cause the death of a living child if any part of that child has been brought forth, though the child may not have breathed or been completely born."

With reference to Explanation 2 of the above section, it may be pointed out that it has been decided in England that when a wound has been given "which in the judgment of competent medical advisers is dangerous, and the treatment which they *bonâ fide* adopt is the immediate cause of death, the party who inflicted the wound is criminally responsible" (*R.* v. *Pym*, 1 Cox, C. C. 339; see Mayne's Penal Code, p. 255). This decision, it will be observed, goes farther than Explanation 2, Sect. 299. Explanation 2 covers all cases where death is due to the effect of neglect or unskilful treatment acting on the injury, but not, like the decision quoted, a case where death is not due to the injury, but to an unnecessary operation undertaken for its cure.

[2] In India the question, has a certain injury caused death, arises irrespective of the period intervening between the receipt of the injury and death. By the law of England, a person is not deemed to have committed homicide if this period exceeds a year and a day (inclusive of the day of receipt of the injury). This provision does not appear in the Indian Penal Code. Mayne, however (Commentaries, p. 265), thinks that it would possibly be acted on in India as a matter of evidence.

2. **Hurt, and grievous hurt.**—According to sect. 319 of the Indian Penal Code, "Whoever causes bodily pain, disease, or infirmity to any person is said to cause hurt;" and by sect. 320, "The following kinds of hurts only are designated as 'grievous': 1. Emasculation. 2. Permanent privation of the sight of either eye. 3. Permanent privation of the hearing of either ear. 4. Privation of any member or joint. 5. Destruction, or permanent impairing, of the powers of any member or joint. 6. Permanent disfiguration of the head or face. 7. Fracture or dislocation of a bone or tooth. 8. Any hurt which endangers life, or which causes the sufferer to be, during the space of twenty days, in severe bodily pain, or unable to follow his ordinary pursuits."

When an act done by another has caused grievous hurt, or hurt, the doer of the act may be charged with the offence of voluntarily causing grievous hurt, or hurt, as the case may be; or according to the circumstances of the case, with the graver offence of attempting to commit murder (sect. 307), or culpable homicide (sect. 308), and causing hurt in such attempt.

Death or hurt caused by a rash or negligent act.—Where a person has caused the death of another by an act not amounting to culpable homicide, he may be charged with the minor offence of causing death by a rash or negligent act (sect. 304 A). Similarly where a person causes hurt or grievous hurt to another, under circumstances which do not amount to voluntarily causing hurt, he may be charged with the minor offence of causing hurt (sect. 377), or grievous hurt (sect. 378), by doing an act "so rashly or negligently as to endanger human life or the personal safety of others." In cases such as these, and in other cases also, besides the main question involved in Question IV., viz., has the injury caused, or is it likely[1] to cause death, grievous hurt, or hurt, a subsidiary medico-legal question may also arise, viz., are the characters of the injury such as to indicate intention, or absence of intention, to cause a particular result? We have now to consider the causes which lead to an injury being followed by a particular result.

[1] It may be remarked that the opinion of a medical expert may be required on the point whether an injury which has caused death is one which comes under the description of an injury "sufficient in the ordinary course of nature to cause death" (Penal Code, sect. 300), or one which comes under the description of an injury "likely to cause death" (sect. 299), *i.e.* whether the injury is one from which death would most probably result, or one from which death would only be a likely result. In the case of *Reg.* v. *Govinda* (1 Bom. 342), Melvill, J., held that under certain circumstances the infliction of an injury which causes death amounts to murder if the injury is one falling within the first of these two descriptions; but only to the minor offence (culpable homicide not amounting to murder) if it falls within the second.

Causes of Death, &c., in Wounds.

1. **Death.**—Some injuries causing death may be called **conditionally mortal injuries**, *i.e.* such as cause death owing to either: (1) **Disease or infirmity** under which the injured individual labours, *e.g.*, an enlarged spleen; or (2) **The supervention of disease**, *e.g.*, tetanus or pyæmia; or, (3) **Want of resort to proper remedies** or treatment, as when death occurs owing to loss of blood from a wounded artery of moderate size, such as the brachial. Others may be called **mortal injuries**, or injuries intrinsically sufficient to cause death, irrespective of the existence of any conditions such as those above mentioned. **Death from a mortal injury** may occur by: (1) **Coma**, *e.g.*, from pressure on the brain of fragments of bone or effused blood; (2) **Asphyxia**, *e.g.*, from paralysis of the movements of respiration, or mechanical interference with this process; (3) **Syncope** from loss of blood, or from mechanical impediment to the heart's action; or (4) **Shock**, as in death from concussion of the brain, or from the effects of a violent blow over the region of the solar plexus.

In some cases, **difficulty may be experienced in tracing the connection between death and an injury** proved or alleged to have been received; thus in the case of injuries alleged to have been caused by the action of external violence on a diseased organ, it may be difficult to determine whether the injury to the organ in question was or was not the result of external violence. In cases of this class, much will depend on (*a*), the liability or otherwise of the affected part to rupture from causes other than externally applied violence (see *post*, *Rupture of the Spleen*, *Injuries to the Brain*, &c.); and (*b*), the presence or absence of marks of violence on the surface of the body over the injured part, or in the tissues situated between it and the surface of the body. Again, when a conditionally mortal injury has, it is alleged, caused death owing to the supervention of disease, it may in some cases be very difficult to decide whether or no the disease is really to be attributed to the injury. **Mortal injuries** causing death by coma, asphyxia, or hæmorrhage leading directly or indirectly to syncope, are not likely to present difficulties of the kind just alluded to. In some cases, however, of death from shock, it is possible that it may be very difficult to trace the connection between death and the alleged violence. With reference to this, it may be pointed out that death from shock may occur: (1) without any mark of violence being present—this has been often observed in cases where the fatal shock has been due to a violent blow over the region of the solar plexus; or (2) a single slight bruise only may be present, as in many reported cases of fatal concussion of the brain; or (3), as often occurs in cases where persons have been severely beaten, from

the combined effect of a number of slight injuries, each by itself totally insufficient to account for death. In cases such as these, it is especially important that the post-mortem examination should be complete, as much may depend on your being able to state (if it be so) that no appearances were present indicative of a cause of death, other than the alleged violence.

2. **Grievous hurt.**—All that is necessary to remark here in regard to this as a result of wounds or mechanical violence is that, in some cases, grievous hurt may follow as an indirect consequence; as, for example, when the injury inflicted sets up inflammatory action, leading to a stiff joint, loss of hearing, &c., &c.

From what has been stated, it is obvious that the results which follow, or are likely to follow from a wound or mechanical injury depend—(1) On the part injured, (2) on the nature and extent of the injury, and (3) on the state of health of the injured individual. Of these factors the first is so important, as to make it necessary to consider separately, the effects likely to follow injuries of each region of the body.

Injuries of Regions.

1. **The Scalp.**—Contused and lacerated, and even apparently incised wounds, are specially likely to follow blows from blunt weapons on the scalp. Occasionally from such blows, the inner surface of the scalp is found ruptured without there being any rupture of the outer surface. Wounds of the scalp only, are not likely to cause danger to life, except from the supervention of inflammation and erysipelas. Other things being equal, these are more likely to follow contused and lacerated, than clean-cut wounds. Erysipelatous inflammation, although a common sequel of scalp wounds in temperate climates, appears to rarely follow such wounds in India.

2. **The Skull.**—Separation of the sutures without fracture, may occur even in old persons, from mechanical violence. Harvey records sixteen cases—one an old man of seventy—in which this was the result of *láthi* blows. Fractures of the skull may be simple or compound, direct or indirect. Simple fracture is a usual result of a fall on a flat surface, while fractures from blows with blunt weapons are, unless the head is protected by a thick turban or some similar covering, usually compound. Fractures from blows with blunt weapons are in the great majority of cases direct, *i.e.*, at the site of the blow. Indirect fracture, *i.e.*, fracture by counterstroke, common as a result of falls, is comparatively rare as a result of blows with weapons. In fractures of the skull, the danger to life mainly depends on the amount of injury to the brain; and other things being equal, the amount of such injury is likely to be greater, the thinner the bones at the seat of

fracture. Hence blows on the temple, and punctured wounds of the orbit, are specially likely to be attended with danger to life.

3. **The Brain.**—Injury to the brain frequently follows a fracture, especially a depressed fracture of the skull, and, as stated above, is the main source of danger in such fractures. Injury to the brain may, however, occur without fracture of the skull, and sometimes results from a comparatively slight blow on the head. As in the case of fracture of the skull by counter-stroke, the seat of the injury to the brain may be at a point opposite to the spot to which the violence was applied. **The brain injury may be of the nature of a contusion followed by concussion.** An injury of this kind may prove immediately fatal, or produce temporary insensibility, which may closely resemble intoxication, and be only distinguishable from it by the absence of alcoholic odour in the breath; or may produce only slight immediate effects, but be followed after an interval by inflammation, ending in death. Guy, for example, mentions the case of a woman who received an injury on the head, and after remaining well for twelve days, fell ill and died with symptoms of compression; and also the case of a girl who, after a fall on the head, suffered simply from headache for six weeks, but died two months after the fall, from brain affection. **Again the brain injury may be of the nature of compression,** caused by depressed bone, effused blood, or the products of inflammation. The brain is specially likely to be injured by **depressed bone,** in punctured fractures, and in fractures in situations where the bones are thin. **Effusion of blood** on the surface, or into the substance of the brain, may occur with or without fracture of the skull, and may cause immediate insensibility, followed by death in a few minutes, or, when the effusion occurs slowly, insensibility may not set in for an hour or more. **Effusion of blood from violence without fracture** of the skull, may or may not be accompanied by appearances of contusion of the integuments covering the skull. If accompanied by such appearances, the question may arise whether the effusion was the result of the external violence which gave rise to these appearances, or the result of disease or excitement. A similar question also may arise even in cases where no marks of external violence are apparent, as effusions of blood from violence may occur without any external signs of injury being present. An effusion of blood from violence is generally, unless the brain itself be torn, on the surface, and not in the substance, of the brain. It is commonly situated immediately below the seat of violence, but in some cases is found at a point directly opposite thereto. **Effusion of blood from disease or excitement** is sometimes extremely difficult to distinguish from effusion caused by violence. **Effusion from disease,** however, rarely occurs in persons under the age of forty; most commonly takes place in

the substance of the brain, and careful examination will generally disclose a diseased condition of the vessels. **Effusion from excitement**—alcoholic or non-alcoholic—may occur in persons of any age. Signs of congestion of the cerebral vessels co-existing with effusion, are to a certain extent in favour of disease or excitement being its cause. It must further be pointed out that even if the probabilities are in favour of an effusion being due to violence, the question may still arise whether the violence was a blow, or the result of a fall. Questions of this kind not infrequently arise in a case of a fight between intoxicated persons. Blows are interchanged; the individuals perhaps are separated; one of them is then seen to stagger and fall, becomes insensible, and dies. Post-mortem examination shows the cause of death to be effusion of blood on the surface, or into the substance of the brain. In such a case it is often difficult in the extreme to arrive at a definite conclusion on the question as to whether the effusion of blood was the result of (*a*) a blow received during the fight, or (*b*) excitement or disease, or (*c*) the fall after the termination of the struggle. **Compression from the products of inflammation** may set in and prove fatal several days or weeks after receipt of the injury.

Lacerations of the brain may be caused by a weapon or projectile penetrating the skull, or by fragments of depressed bone; or may occur without injury to the skull, either immediately below, or at a point directly opposite to, the seat of the violence. Wounds of the brain are of course attended by great danger to life. Very severe wounds of the brain, accompanied even by loss of substance, may not cause immediate death, or even immediate insensibility (see *Question III.* 3, p. 51), and in exceptional cases recovery may take place.

4. **The Face.**—Wounds of the face are not likely to be dangerous to life unless the injury or resulting inflammation extends to the brain. Injuries to the face by causing permanent disfiguration, loss of sight, &c., often come within the definition of "grievous hurt." Wounds, with or without partial removal, of the nose and ears, are often met with in India, the motive leading to the infliction of the injury being usually sexual in character. This is specially so when the victim is a female, and a sharp-edged weapon has been used. Often when the victim is a female the lips or breasts are also wounded, and often also no other injury is present, indicating either submission of the sufferer to the punishment, or the participation of several persons in the outrage. When the victim is a male the motive is commonly either sexual, or punishment for theft; or, if the teeth have been employed, the injury may have been inflicted in the course of a struggle, and indicate no special motive. Injuries to the nose and ears caused by forcibly pulling out ornaments are

not uncommon, especially in females, and may, by causing permanent disfiguration, amount to grievous hurt. In such cases the motive may either be theft, or desire to cause hurt. Injuries to the eyes also are not uncommon, and may be the result of direct violence, *e.g.*, gouging out by the fingers, or injury by a sharp-pointed weapon, the motive for infliction of the injury being similar to those leading to wounds of the nose or ears. Or the injury may be the result of indirect violence, and indicate no special motive. As examples of injury to the eyes from indirect violence, it may be mentioned that blows with a club on the head, sometimes cause rupture of the eyeball; and wounds of the eyebrows, are sometimes followed by amaurosis.

5. **The Spine and Spinal Cord.**—Serious injuries to the cord above the third cervical vertebra, are immediately fatal from paralysis of the muscles of respiration. Serious injuries lower down give rise to secondary effects, from which death may follow long after the receipt of the injury. Injury to the spinal cord may occur without fracture or dislocation of the vertebræ. A blow, for example, on the spine, may cause concussion of the cord followed by paralysis, or may set up inflammation followed by softening of the cord. Concussion of the cord sometimes results from a railway accident, and in actions for damages in cases where this injury is alleged to have been received, the question whether the plaintiff's symptoms resulted from the accident or from disease, is sometimes a very difficult one to deal with. Fracture of the second cervical vertebra with displacement and immediate death, is a not infrequent result of a fall from a height on the vertex. If the bones or ligaments are diseased, very slight violence may cause displacement and fatal injury to the cord, and Taylor mentions a case in which displacement of the odontoid process, and fatal injury to the cord, appear to have been caused simply by the muscular effort of throwing the head forcibly back. Fatal injury to the cord from non-accidental violence, may be caused without a weapon. Harvey, among the medico-legal cases reported in Bengal, &c., during the three years ending 1872, mentions three in which violence used in wrestling resulted in fracture of one of the cervical vertebræ and death. In two of these cases the third and in one the fifth cervical vertebra was fractured. The same author mentions a case in which fatal injury to the cord in a boy æt. six, resulted from dislocation of the sixth from the seventh cervical vertebra caused by twisting the neck; in this case no external marks of violence were present on the body. Fatal injury to the cord, unaccompanied by injury to any portion of the body other than the spine, is rare as a result of blows from blunt weapons, but may occur when the neck is the seat of the injury, and may even occur without any external marks of violence being present. In one of Harvey's cases, for example, a woman æt. sixty was killed

by a blow with a club on the neck. Death resulted from injury to the cord due to displacement of the vertebræ, but no external marks of violence could be seen, although on dissection blood was found effused into the muscles of the nape. Hacking the spine with a sword, bill-hook, or other heavy cutting weapon—causing sometimes decapitation—is, Harvey points out, a common mode of murder all over India, and specially so in the Central Provinces, Oudh, and the Punjaub.

6. **The Neck.** — Injuries of this region from mechanical violence other than the use of edged weapons, are chiefly dangerous to life from their effect on the spinal cord. A case cited by Harvey, however, shows that mechanical violence may cause very extensive, possibly fatal, injury to the soft parts in front of the neck without dividing the skin. **Wounds of the neck from edged weapons** are often suicidal, and often also homicidal. Suicide is more or less contra-indicated, if the wounds are multiple, unless one only is severe; or if the wound is single and of great severity, more than sufficient to destroy life; or if the wound is low down on the neck (see also pp. 49 and 51). Wounds of the neck vary in danger to life according to their situation and depth. From the position of the large blood-vessels lateral wounds are more dangerous to life than wounds in front, and wounds low down on the neck more dangerous than wounds high up. Wounds of the larynx or trachea only, are attended with little danger to life. Wounds of the neck dividing the œsophagus are almost always fatal. Wounds of the large vessels are mortal injuries, death resulting either from hæmorrhage, or from the entry of air into the circulation. Wounds of the carotids are not necessarily immediately fatal (see p. 53).

7. **The Thorax.** — **Penetrating wounds** of the thorax, perforating the heart or one of the large vessels, are mortal, but not necessarily immediately mortal, wounds. In such wounds the rapidity with which death occurs, greatly depends on the rapidity with which hæmorrhage takes place. Loss of blood may occur comparatively slowly if a large vessel is only punctured, and the puncture is small; or if the heart is wounded, if the wound is small, or oblique in direction. After a wound of the heart an individual may even survive several days. Taylor mentions two cases, one of survival for eleven days with a bullet one-third of an inch in diameter, lodged in the septum between the ventricles; and another of survival for five weeks with a mass of wood, lodged in the substance of the heart. Even when death occurs rapidly, considerable power of locomotion may remain after receipt of a wound of the heart, as in the case already mentioned, where a man ran eighteen yards after a stab penetrating the right ventricle. Taylor also mentions a case in which it is probable that a man ran over eighteen feet after a gunshot wound "shattering to atoms" the auricles and part of

the aorta. If the lungs are wounded, death may occur rapidly from hæmorrhage, or after an interval from inflammation, but wounds of the lungs are not necessarily mortal wounds. A wound completely transfixing the chest, other things being equal, is not more dangerous than a simple penetrating wound.

Non-penetrating wounds and injuries of the thorax are dangerous to life in proportion to the amount of internal injury. Serious internal injuries of this class are usually, but not invariably, accompanied by fractures of the ribs, but fractures of the ribs may be present without other internal injury. If a rib has been fractured by direct violence, *e.g.*, a blow from a blunt weapon, it is usually found broken in one place only, and the ends are driven inwards. When the fracture has been the result of indirect violence, the broken ends are usually driven outwards, and the fracture, if single, is generally at the point of greatest convexity. Ribs when fractured by indirect violence are often broken in two places, one in front and the other behind. Very often also when the violence is of the nature of a force compressing the thorax, the fractures are symmetrical or nearly so; *i.e.* fracture of a rib on one side of the body, is accompanied by fracture of the corresponding rib on the other side. **Compression of the thorax**, causing symmetrical indirect fractures of the ribs, may be due to accidental violence, *e.g.*, the fall of a heavy weight on the front of the chest, or more rarely to a fall from a height. More frequently it is the result of homicidal violence, and may be due to pressure with the knees, trampling under foot, or to compression of the body between two bamboos, a process known as "*bans-dola.*" Again, it may be due to kneading with the knees and elbows or "*kíl kani*" (see also injuries to the liver). Harvey mentions a case in which symmetrical rib fractures were present, but no external marks of injury were to be seen on the chest, and suggests that in the case in question the compressing force was probably pressure with the knees.

Non-penetrating injuries of the thorax may be accompanied by injury to the lungs or heart. If the lungs are injured, hæmothorax or inflammation, either of them ending fatally, may follow, even when there is no fracture of the ribs. Emphysema may be present, but this is only dangerous to life from mechanical impediment to respiration.

Rupture of the heart is a comparatively rare result of non-penetrating chest injuries. Harvey, however, mentions fourteen cases, five of them homicidal, in which it occurred. Rupture of the heart may occur solely from disease, or from a comparatively slight amount of violence acting on a diseased heart. Hence, when the heart is found ruptured and no marks, or slight marks only, of external violence are present, it may be difficult to decide whether or no the rupture was due solely to disease. Non-penetrating chest injuries may cause rupture of a large thoracic

blood-vessel, *e.g.*, as in cases mentioned by Harvey, of the pulmonary artery, pulmonary veins, or superior vena cava. Rupture of the diaphragm also may occur, followed by hernia, liable to undergo strangulation, and thus cause death long after the receipt of the injury.

8. **The Abdomen.**—**Penetrating wounds** unaccompanied by any internal injury are, even if accompanied by protrusion of viscera, not necessarily fatal. Death, however, may occur rapidly from shock, or after an interval from peritonitis. Moreover such wounds, and also wounds or rupture of the diaphragm, are liable to be followed by hernia, and may hence cause death indirectly, after a long interval. An internal injury accompanying a penetrating wound of the abdomen, may be a wound of a vascular organ or large vessel leading to death from hæmorrhage; or a hollow viscus may be wounded and extravasation of its contents be followed by fatal peritonitis. **Fatal non-penetrating injuries** of the abdomen may leave no external marks of violence. In some, but not all such cases, the tissues immediately underlying the skin at the seat of injury, may on dissection be found to show signs of bruising, and to contain extravasated blood. Blood however, it must be recollected, may in rare cases be found extravasated in the muscles of the abdominal wall, without violence having been applied. Taylor[1] mentions two such cases; in both the extravasation was into the muscles around the navel. **A non-penetrating injury unaccompanied by any wound of the contents** of the abdominal cavity, may cause immediate death from shock. This is specially liable to occur from a blow over the region of the solar plexus, and in such a case, after death, no marks of violence, external or internal, may be discoverable. Or an injury of this class may cause death from peritonitis, in which case after death, no lesion other than signs of inflammation of the peritoneum may be found. More frequently the cause of death in fatal non-penetrating abdominal injuries is rupture of a viscus such as the spleen or liver. **Rupture of a viscus**, however, it must be recollected, may occur from post-mortem violence, especially when decomposition is far advanced. An idea of the relative frequency of occurrence in India of rupture from violence of the different abdominal viscera may be gathered from the following figures. Harvey among the fatal medico-legal cases reported in Bengal, &c., during the three years ending 1872, records rupture of the spleen in 564, liver in 129, bowels in 25, kidney in 24, urinary bladder in 8, and of the stomach in four or five cases.

The Spleen.—Rupture of this viscus is only recovered from in very exceptional cases. Disease of the spleen,[2] rendering it

[1] Medical Jurisprudence, i. p. 647.

[2] McLeod, quoted by Chevers (p. 462), points out that rupture of the spleen is liable to occur in cases of (1), simple engorged spleen; (2) hypertrophied engorged spleen; (3) small hard spleen; (4) large hard spleen.

liable to rupture from slight violence is, as is well known, very common in India. Rupture of the spleen—probably, however, only when the organ is in an advanced state of disease—may even occur during life without the application of external violence. So slight often is the force required to rupture a diseased spleen, that in many cases where this occurs from violence inflicted by another, there is no intention of causing death. Rupture of the spleen may be due to **accidental violence**, *e.g.*, a fall, or from the sufferer having been run over by a wheeled vehicle. **In non-accidental** cases it is often the result of a blow without a weapon. It may occur without any external marks of violence being present—this was so in about one-third of Harvey's cases—but in about one-fifth of these the tissues under the skin over the region of the spleen, on dissection, showed signs of bruising. Rupture even of an apparently healthy spleen, may be unaccompanied by external marks of violence, but in such cases the subcutaneous tissues will probably (but not certainly) show signs of bruising. Death may occur in a few minutes or not for several days. Chevers mentions one case of survival for five days, and another of death on the eighteenth day from pleurisy and pericarditis. Considerable power of locomotion may remain after receipt of the injury. Harvey mentions a case in which a man whose spleen was extensively ruptured walked two miles (see also *Case* A 16).

The Liver.—There is reason to suppose that in very exceptional cases, recovery may take place after a slight rupture of this viscus, and also that in very exceptional cases, rupture of the liver may occur during life, without application of external violence. Non-accidental rupture of the liver may be caused without a weapon. Harvey, for example, mentions a case where it was ruptured by a kick, and two others in which the rupture was caused by kneading with the knees and elbows, or "*kil kani*" (see also *ante*, p. 63). Rupture of the liver may occur from violence inflicted during life without any external marks of injury being left. In about one-fifth of Harvey's cases no external marks of injury were present. Case A 16 shows that considerable power of locomotion may remain after receipt of the injury. Taylor remarks, that unless the large veins at the back of the liver are injured, bleeding from a ruptured liver may occur only slowly, and the patient survive some time; but thereafter die rapidly from sudden copious effusion of blood, caused by muscular exertion, or fresh violence. The same author mentions one case of survival for eight days, and two of survival for ten days, after rupture of the liver. **The gall bladder** may be ruptured by violence, as in a case mentioned by Harvey in which the subject was a boy æt. five, who had been strangled, and in which the rupture was probably caused by pressure with the knees. Ogston, however, remarks that "ruptures of the gall bladder proper have usually

been the result of emetics given to ensure the expulsion of gall stones."

The Intestines.—Rupture of the intestines is usually fatal, the cause of death being commonly peritonitis, the result of extravasation of their contents. Rupture may occur solely from disease, or from violence acting on a diseased portion of the intestine, or solely from violence. Hence, when this injury is found, careful examination of the ruptured portion for signs of disease, *e.g.*, ulceration or softening, is of special importance. Rupture even of a healthy portion of the intestines, may occur from a comparatively slight amount of violence. The violence causing the rupture may leave no external marks. Out of Harvey's twenty-five cases, in twelve, external marks were absent; but in five of these, on dissection, signs of bruising were found in the subcutaneous tissues. Rupture of the intestines may be the result of accidental or non-accidental violence, seven of Harvey's cases apparently were accidental. When non-accidental, the injury is often the result of a blow without a weapon; usually after the receipt of the injury, the sufferer is capable of considerable muscular exertion. (*For injuries to the rectum*, see p. 69.)

The Stomach.—This viscus also is liable to rupture from disease. Cases also are recorded of rupture from over-distension and violent ineffectual efforts to vomit, and of spontaneous rupture without any very apparent cause. Taylor mentions a case in which rupture both of the stomach and the spleen occurred from a fall of about twenty feet, and in which no bruises or other external signs of injury were present. In one of Harvey's cases, also, although there was a fracture of the skull, and bruises on various parts of the body, the result of *láthi* blows, no external sign of injury could be found over the region of the stomach, although this viscus was ruptured. It is possible, therefore, that rupture of the stomach from accidental or non-accidental violence may occur, and no external signs of injury be present. **The Pancreas.**—Injury to this viscus from external violence is very rare. McLeod and Harvey, however, each mention a case; in the first the viscus was ruptured but no external marks of injury were present; in the second the viscus was "injured," and contusions, not visible externally, were present on both sides of the spine. In both, the injury appears to have been caused by kicks or trampling with the feet.

The Kidneys.—Rupture of the kidney solely from disease is extremely rare. Disease or abnormal formation of this viscus may, however, conduce to rupture from violence. Rupture of the kidney usually occurs only from great violence, and hence is often accompanied by other lesions. Harvey remarks that, notwithstanding this, in nearly one-half of his cases, signs of external violence over the region of the kidneys were absent. In sixteen of Harvey's cases, the nature of the violence causing

the rupture was stated. This was in eight, blows from blunt weapons; in two, kicks; in one, trampling; and five resulted from falls from a height. Taylor mentions a case in which a man walked some distance after an accident whereby one kidney was torn completely across, death occurring suddenly, within six hours after receipt of the injury.

The Bladder.—In rare cases, rupture of the bladder occurs solely from disease, either of the bladder itself; or disease, *e.g.*, paralysis or stricture, leading to over-distension. In the great majority of cases, however, the cause of the rupture is violence applied directly to the region of the distended organ. Often in cases of rupture from violence, no external marks of injury are to be found. The injury is usually fatal, either from shock, or peritonitis due to extravasation of urine; recovery, however, sometimes takes place. The violence leading to the rupture may be accidental, *e.g.*, a fall from a height, or a fall on some projecting object; or non-accidental, *e.g.*, a kick in the pubic region. In females, rupture of the bladder sometimes occurs from pressure of the child's head during delivery.

The Uterus.—Rupture of the unimpregnated uterus is only likely to occur from very great violence. Rupture of the pregnant uterus may occur independently of violence, as an accident during delivery, and, in rare cases, may be partial only, affecting the peritoneal coat and muscular tissue, but not extending into the cavity. Rupture of the pregnant uterus may occur from external violence without any external marks of injury being present. The uterus often apparently escapes injury, even when great violence is applied to the abdominal wall in attempts to cause miscarriage (see *Cases* C 42–44). Harvey, however, mentions a case in which extensive bruising —not rupture—of the uterus caused in this way, resulted in death; in this case also, no external marks of violence were present. The uterus may be wounded per vaginam, in an attempt to cause miscarriage (see p. 376). Injury to the uterus per vaginam, also sometimes results from thrusting sticks, &c., into the vagina after rape, or in revenge for infidelity. Rupture of an ovary, or fallopian tube, may be found; this, it must be remembered, may occur as a result of ovarian or tubal pregnancy, and hence blood-clots, &c., found in the abdominal cavity should in such cases be carefully searched for an embryo.

The large abdominal blood-vessels.—Harvey mentions three cases of rupture of the inferior vena cava from external violence. In one—caused by the sufferer being jammed between two boats—bruising of the mesentery was the only other serious injury present. In another—the result of a fall from a high tree—the skull was also fractured; and in the third, the liver was ruptured. The same author also mentions a case of probable rupture of the splenic vein.

9. **The External Genitals.**—(*a*) **The male genitals.**—Severe contusions, or severe compression of the testicles may prove fatal from shock (see *Case* A 20). Seizing by the testicles is a common method of assault in India, and Chevers mentions a case in which a man dragged another along in this way with such violence "that the whole preputial integument was torn away." Incised wounds may be attended with severe and even fatal hæmorrhage, or by extravasation of urine, ultimately terminating fatally. Incised wounds, even removing the whole of the external genitals, are sometimes self-inflicted; generally, however, in such cases the individual is insane, but Harvey mentions one case and Chevers another, where individuals apparently perfectly sane, mutilated themselves by cutting off a considerable portion of the penis. In India, removal of the male genital organs used formerly to be largely practised, in order to manufacture eunuchs for immoral purposes. Young boys were generally selected, and a clean sweep made of the whole of the organs. Chevers, on the authority of Dr. Ebden, appears to consider that, in 1870, this practice still existed extensively in the Rajputana States, and Harvey (1870–72) mentions the case of "a Chamar boy, aged eight, at Banisal, whose genitals were completely cut away, probably to fit him for the duties of a eunuch." Cases of this kind excluded, incised wounds of the male genitals inflicted by another, indicate, as a rule, a sexual motive. Occasionally, the person inflicting the injury is a female, as in a case cited by Harvey, of a woman at Kachar, who "seized a weapon and inflicted a deep and severe wound on the penis of her father-in-law, who wished to take liberties with her." Harvey also mentions an exceptional case, in which a eunuch possessed of a penis, had it shaved off by some of his fellow-eunuchs, apparently from motives of jealousy.

(*b*) **The female genitals.**—Incised, or even contused, wounds of these may prove fatal from loss of blood. Some years ago, several cases occurred in Scotland of murder by wounding the female genitals. In one of these, death occurred in ten minutes; and in another, a wound of the labium three-quarters of an inch long and three inches deep, proved rapidly fatal from loss of blood. A kick on the vulva—like a blow on the head—may cause an apparently incised wound, and prove fatal from hæmorrhage (see *Case* A 21). Fatal hæmorrhage may, however, occur without external violence, from spontaneous rupture of a large vein in one of the labia, as in a case referred to by Ogston.[1] Wounds of the female genitals are sometimes the result of an accidental fall on some projecting sharp or pointed object. In India, cases of injury by thrusting a stick into the vagina are not uncommon. Harvey states, that twenty-five such cases, ten of them fatal, were included in the Bengal, &c., returns

[1] Med. Jurisp., p. 486.

for 1870–72. Sometimes such injuries are inflicted after rape has been committed. Similar injuries are sometimes produced in attempts to procure abortion (see p. 376).

10. **The Rectum.**—Thrusting a stick or other similar object into the anus, is a mode of torture or murder, occasionally resorted to in India. Injuries produced in this way may cause death. Fifteen cases—eleven of them fatal—of the infliction of this form of violence were included in the three years' returns for Bengal, &c., reported on by Harvey. Very often other injuries accompany this form of violence. An individual, for example, is attacked and violently beaten by several others, and finally thrown down and subjected to it. In the majority of such assaults, the victim is a male, and the motive leading to the infliction of the injuries appears to be most commonly punishment for adultery or theft. Possibly, also, in some cases, the injury is connected with sodomy, in the same way as similar injuries to the vagina are sometimes connected with rape. Chevers mentions a case in which several children of about eight or nine years old, threw down a boy, one of their number, and killed him by thrusting a small stick into his rectum, and Harvey mentions a similar case, said to have been accidental. Harvey points out that dilatation of the rectum, and protrusion of the gut, is a common effect of putrefaction, and hence that such a condition does not necessarily indicate the infliction of this form of injury. Injuries to the rectum and anus are sometimes the result of an act of sodomy (see *Evidence of Sodomy*, p. 330).

11. **The Extremities.**—Injuries to the extremities vary greatly in gravity, according to their situation and extent. Death may result if a large vessel is wounded, directly from loss of blood, or, if the injury is severe, from shock; and slight injuries may contribute to the production of fatal shock in cases where this results from numerous slight injuries. Again, injuries to the extremities may prove indirectly fatal from inflammation and exhaustion, or from supervention of disease; or, if the large vessels or nerves are divided, may necessitate amputation, followed by similar consequences. Injuries to the extremities necessitating amputation, or permanently impairing their power, of course amount to grievous hurt. Obviously, injuries to the extremities may be accidental, or self-inflicted. No further remarks are called for here in regard to these. As regards injuries inflicted by another, it may be pointed out that very severe injuries of the extremities may be produced without a weapon. Violent twisting of a limb, for example, may cause dislocation of a joint. Again, comparatively slight injuries to the extremities, especially when caused by ropes or cords, may indicate the infliction of very severe torture. The nature of the torture thus indicated may be: torture by compression, as when the fingers are tied together and wedges driven in between them,

or torture by binding the body or limbs in a constrained position, or torture by suspension. Harvey mentions a case which shows that suspension head downwards may cause death from congestion of the brain. Cases in which the right hand is completely severed from the body are tolerably often met with in India. This, especially if accompanied by mutilating wounds of the ears or nose, usually indicates punishment for adultery or for theft, as the motive leading to the infliction of the injury.

Detection of Blood and identification of fibres found adhering to weapons, &c.

Blood dried on weapons, articles of clothing, &c., is recognized by **A. The reactions of its colouring matter,** and **B. The identification of blood corpuscles** under the microscope.

A. The reactions of the colouring matter of blood include: 1. Chemical, and 2. Spectroscopic, reactions. **The chemical reactions** are as follows:—

(1) It strikes a sapphire blue colour, with guaiacum resin in presence of hydrogen dioxide.

(2) It dissolves in cold water, yielding a red solution.

(3) Its solution, on being heated, coagulates and loses its colour.

(4) Its solution, on addition of a drop or two of nitric acid, coagulates and loses its colour.

(5) Its solution, on addition of dilute ammonia, either remains unaltered, or becomes bright red in tint.

(6) If the residue obtained on evaporating a solution of blood to dryness is boiled with the strongest glacial acetic acid, and a drop of the acetic acid solution is allowed to evaporate spontaneously, minute crystals (hæmatin crystals) form, visible under the microscope.

As regards the above reactions—

(1) Is applied as follows. The stain is first moistened with freshly-prepared alcoholic tincture of guaiacum resin, and then a few drops of ethereal solution of hydrogen dioxide (ozonized ether) are added; if the stain is of blood, a bright sapphire blue colour rapidly appears. Should the stained fabric or object be so dark in colour as to obscure the reaction, press pieces of white filter paper on the stain after the reagents have been applied. The blue colour, if the stain is of blood, will appear on the paper. Many matters, however, other than blood, give this reaction, and hence the guaiacum and ether test by itself, cannot be relied on as positive evidence of the presence of blood. The test, however, is useful as a preliminary test, as stains which do not give this reaction cannot be stains of blood.

(2) If the stains are on cloth, or some other similar fabric, suspend a portion of the stained article by a thread, in a little

distilled water contained in a test-tube. The water, especially near the bottom of the tube, soon acquires a red colour. This distinguishes blood from various fixed red dyes. The colouring matter of blood, however, may be rendered insoluble by the action of various substances, *e.g.*, tannic acid and iron rust; hence blood stains on leather, and old blood stains on iron weapons, may not yield a red solution to water. Heat also, and age even, may render the colouring matter of blood insoluble in pure water. In such cases dilute solution of ammonia, or dilute solution of citric acid, or, if the stain is on leather, dilute hydrochloric acid, should be used as a solvent in place of water.

(3) Soluble red colouring matters other than blood do not coagulate and lose their colour on the application of heat. A mixture of a soluble red colouring matter with albuminous matters will coagulate on being heated, but will not lose its colour. The coagulum obtained by heat from blood is soluble in potassic hydrate solution, forming a liquid green by reflected, but red by transmitted light.

(4) and (5) The majority of red colouring matters, by the action of ammonia, change either to blue or green, *e.g.*, fruit and flower stains, or to crimson, *e.g.*, Brazil wood; or lose their colour, *e.g.*, sulphocyanide of iron and rosaniline. A few are not changed by ammonia, but most of these either turn yellow on the addition of acids; or, like catechu and kino, give, owing to the presence of tannic acid, a blue-black or dark green precipitate or colour, with iron salts. It should be noted that the coagulum produced in blood by dilute nitric acid, is dissolved by hot strong nitric acid.

(6) The hæmatin or blood-crystals obtained by this process are minute, and require for recognition of their form a magnifying power of 300 to 500 diameters. They are of various shapes, generally prismatic or rhomboidal. A minute quantity of common salt may be added to the acetic acid solution, or a dilute solution of common salt, or a solution of potassium iodide (1 to 4 Falk), may be used instead of water, for the purpose of dissolving the stain. If these salts are employed, the cubical crystals, which their solutions yield on evaporation, must not be mistaken for hæmatin crystals.

2. **Spectroscopic Reactions.**—A solution of blood examined by the spectroscope shows the following spectra:—

(1) If the blood is tolerably fresh, the spectrum of **oxidised hæmoglobin**, characterized by two absorption bands in the green.

(2) If the blood is not fresh, the spectrum of **methæmoglobin**, viz., two faint absorption bands in the green, accompanied by a third band in the red; this last disappears on the addition of ammonia.

(3) On addition of reducing agents to the blood, the spectrum

of **deoxidised hæmoglobin**, characterized by a single absorption band in the green.

(4) After free exposure of the reduced solution to the air the spectrum of deoxidised hæmoglobin is replaced by that of oxidised hæmoglobin.

In all the above spectra, the blue end of the spectrum is more or less darkened.

For observation of blood spectra it is convenient to employ a microspectroscope, and to use, for the purpose of holding the solution under examination, a small cell composed of a piece of barometer tubing, about half an inch in length, fixed on a piece of glass. Or a cell may be used, made of about half an inch of the closed end of a very narrow test tube, inserted into a phial cork fixed in a circular aperture in a piece of wood, shaped like a microscope slide. The spectrum of the liquid under examination should be compared with that of a solution known to be of blood. For this purpose microspectroscopes are fitted with an arrangement whereby the spectrum yielded by a comparison liquid, can be observed simultaneously with that of the suspected fluid. The reducing agent for reaction (3) may be either ammonium sulphide or a minute quantity of ammonia, followed by a minute fragment of Rochelle salt, and subsequently by a minute fragment of ferrous ammonium sulphate. If citric acid is substituted for Rochelle salt in reaction (3), the spectrum of reduced hæmatin is obtained, viz., two dark bands in the green.

B. The identification of blood corpuscles under the microscope. These in shape are—

(1) **Circular biconcave discs** in human blood (see Fig. 1), and in the blood of all mammalia except the camel tribe.

(2) **Oval biconcave discs** in the camel tribe.

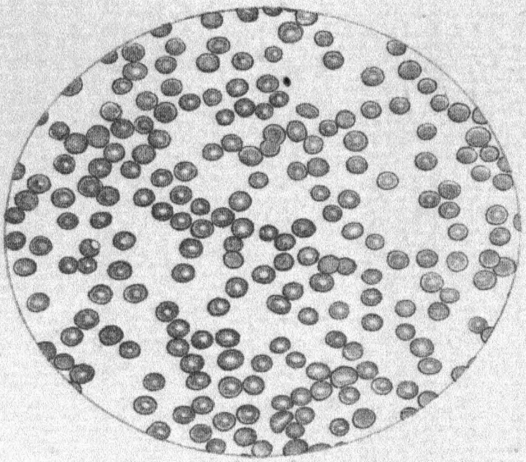
(3) **Oval nucleated discs** in birds, fishes, and reptiles (see Fig. 2).

The corpuscles vary in size in the same animal, and in average size in different animals. In man they range in diameter from $\frac{1}{2800}$ to $\frac{1}{4000}$ of an inch, averaging $\frac{1}{3200}$. The average diameter of the blood corpuscles of the following mammalia ranges from about $\frac{1}{3400}$ to $\frac{1}{4000}$ of an inch—the animals are named in order of the size of their corpuscles, those with the largest first—the monkey, dog, hare, rabbit, rat, mouse, and bat. In the following animals enumerated in similar order, the average diameter of the corpuscles ranges from about $\frac{1}{4000}$ to $\frac{1}{4600}$ of an inch—the ass, pig, ox, cat, and horse. In the sheep and goat they average less than $\frac{1}{5000}$, and in deer less than $\frac{1}{7000}$ of an inch in diameter.

Blood corpuscles are liable to alter in size from imbibition or exudation of fluid; hence, in removing them from a stain for examination, a liquid should be employed of nearly the same specific gravity as blood serum. One volume of glycerine to six or seven of water, is a suitable and convenient fluid for this purpose.

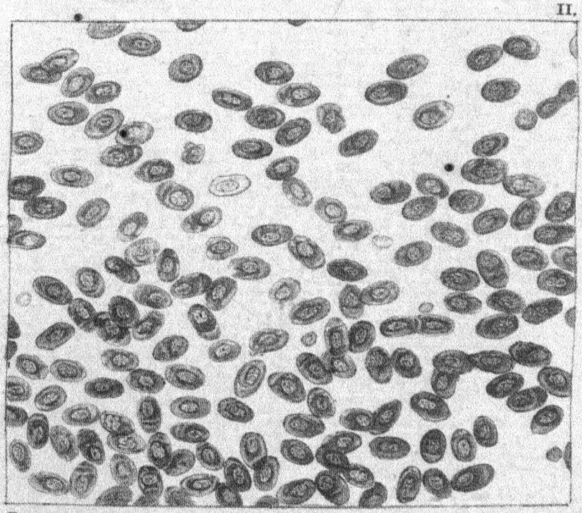
The Measurement of blood corpuscles requires (*a*) a stage micrometer, *i.e.*, a glass slide on which are engraved lines a given distance—say some $\frac{1}{100}$

I.



HUMAN BLOOD × 320.

II.



Page 72.

AMPHIBEAN BLOOD × 130.

From Micro-Photographs by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

BENGAL SECRETARIAT
LIBRARY

and others $\frac{1}{1000}$ of an inch—apart, and (*b*) an eye-piece micrometer, *i.e.*, an eye-piece fitted with a disc of glass, on which a scale is engraved. The procedure is as follows: first place the stage micrometer on the microscope stage, and examine it under the microscope, using the eye-piece micrometer. Note the number of divisions of the scale of the eye piece micrometer, which appear included between two of the $\frac{1}{1000}$ of an inch apart lines of the stage micrometer; say, for example, four are included, then each division of the scale of the eye-piece micrometer corresponds, for the power in use, to one-fourth of one thousandth, or $\frac{1}{4000}$ of an inch. Next remove the stage micrometer, substitute the slide containing the corpuscles to be measured, view these with the aid of the eye-piece micrometer, and measure them by bringing them between the lines of its scale. Instead of an eye-piece micrometer a camera lucida may be employed. In this case the divisions of the scale of the stage micrometer are first drawn on paper with the aid of the camera. Then the blood corpuscles are similarly drawn, of course using the same magnifying power, and their size ascertained by comparison of the two drawings.

The following points are to be noted in regard to the detection of blood:—

1. **Recently effused blood,** if seen before it has become dry, is so peculiar in colour, consistence, and general appearance, that it may be identified by these characters alone, by any ordinary unskilled observer.

2. **Very recent stains of blood** dissolve readily, their solution giving the spectrum of oxidised hæmoglobin. Old stains dissolve less readily, and give the three-band spectrum of methæmoglobin. If a stain possesses the first-mentioned characters, it may be declared to be a comparatively recent stain; if it does not, it may be declared to be not a very fresh stain, but no further opinion can be given as to its age.

3. **Blood stains** may be completely **removed** by efficient washing with cold water.

4. **Human blood, once it has become dried, cannot be distinguished** from the blood of many other animals of the class mammalia. An opinion on this point, founded on the size of the corpuscles, is not reliable. Mammalian blood, by the shape of its corpuscles, can of course be distinguished from the blood of birds, fishes, and reptiles.

5. **Menstrual blood** only differs from other blood in (*a*) containing no fibrin, and (*b*) being mixed with scaly epithelium derived from the vaginal walls. Hence, if on examination of a stain under the microscope, no scaly epithelium is found mixed with the blood, the probabilities are great that it is not menstrual blood. On the other hand, should the stain contain both scaly epithelium and blood, an opinion may be given to the effect, that the appearances of the stain are consistent with the supposition of its being a stain of menstrual blood. In no case, from examination of a dried stain, can it be positively asserted that the blood detected is of menstrual origin.

Identification of fibres found adherent to weapons, &c.

The fibres more commonly found adherent to weapons when examined under the microscope exhibit the following characters:—

Silk fibres are cylindrical, transparent, refract light powerfully, and are free, or almost free, from surface markings (see Fig. 3).

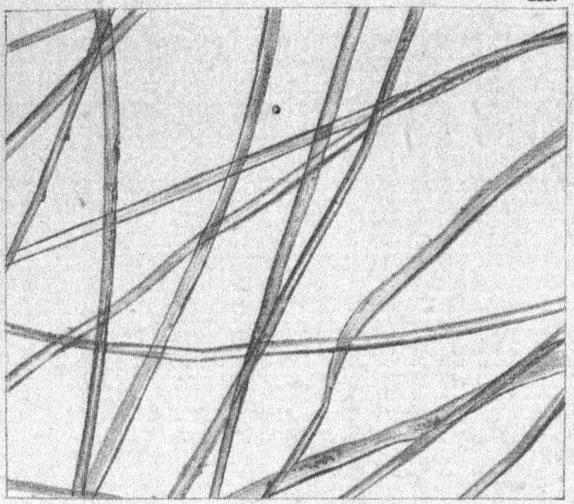
Cotton fibres are flattened, transparent bands. Each band is twisted once on itself at intervals equal in length to about seven or eight times the breadth of the band (see Fig. 4).

Linen fibres are cylindrical, transparent, and apparently jointed at intervals something like a bamboo (see Fig. 5).

Wool fibres (really animal hairs) are cylindrical, and marked on the surface, at close intervals, with fine, but very distinct, oblique cross-markings, the remains of a cortical layer of large imbricated scales (see Fig. 6).

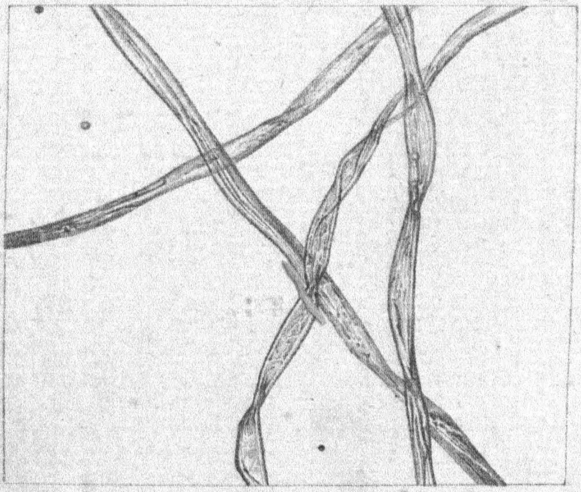
Human hairs (see Fig. 7*a*) are cylindrical, and marked on the surface with very fine transverse cross-markings, of the same nature as those present on wool fibres, but much finer. The cortical layer is extremely thin, and the imbricated scales composing it, indicated by the cross-markings, are much smaller in proportion to the width of the hair than is the case in the hair of many of the lower animals. Sometimes in human hairs a narrow axial band of spheroidal cells is visible: this band is broader, and much more distinct, in many varieties of non-human hair. The hair of some of the lower animals, *e.g.*, of a brown dog, very closely resembles human hair. When examining a specimen of hair supposed to be human, it is always advisable to compare it with an authentic specimen of human hair, taken, if possible, from the individual whose body is supposed to have furnished the hair under examination. The condition of the ends of the hairs under examination should be noted, *i.e.*, whether one or both ends appear cut, or whether one end shows a point and the other the bulbous root of the hair. Figs. 7*b* and 7*c* show the appearance under the microscope of the hair of the cat and guinea pig.

III.



SILK FIBRE × 130.

IV.



COTTON FIBRE × 130.

From Micro-Photographs by Dr. Heneage Gibbes.

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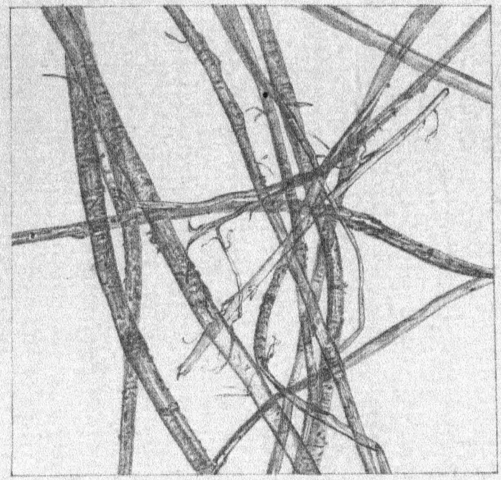
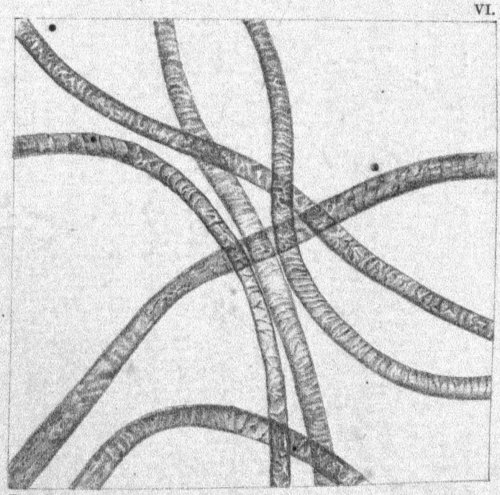
V.



LINEN FIBRE × 130.

VI.

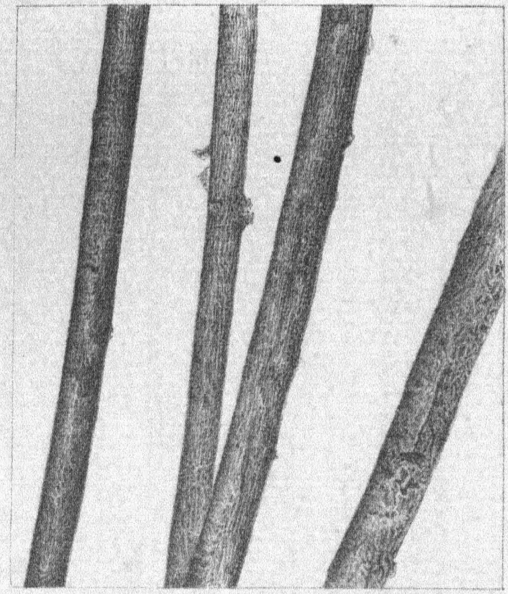


Page 74. SHEEP'S WOOL × 130.

From Micro-Photographs by Dr. Heneage Gibbes.

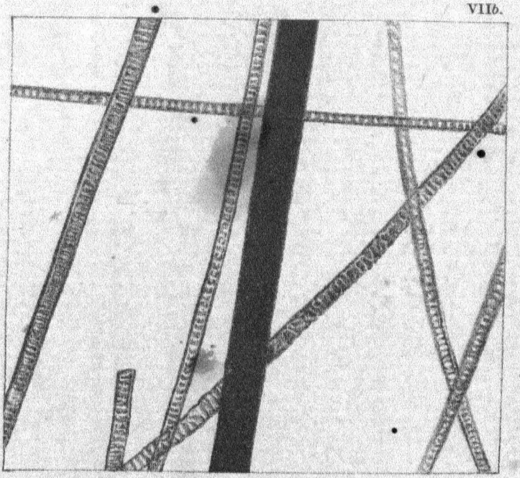
Thacker, Spink, & Co., Calcutta.

VII*a*.



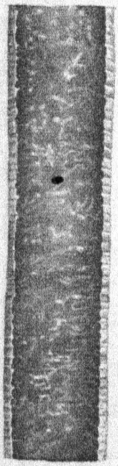
HUMAN HAIR × 130.

VII*b*.



CAT'S HAIR × 130.

VII*c*.



HAIR OF GUINEA PIG × 130.

From Micro-Photographs by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

CHAPTER IV.

SUDDEN DEATH WHERE APNŒA IS THE GENERAL CAUSE, VIZ., DROWNING, HANGING, STRANGULATION, AND SUFFOCATION.

DROWNING.

Origin of cases.—**Accidental cases** are common in all countries among the seafaring population of the coast. Accidental cases also are common throughout India generally, especially among females, from falls into wells and tanks. In the Bombay Presidency in 1883 (see Table VIII, *Appendix*, p. 497) nearly one-half of the violent deaths of the year were cases of drowning, and of these over three-fourths were accidental. **Suicidal cases** are also common in India. In the Madras and Bombay Presidencies (see Table VII, *Appendix*, p. 496), over three-fourths of the female, and nearly one-half of the male suicides, drown themselves. In European countries also, drowning is a mode of suicide very commonly selected, ranking, as a rule, second in order of frequency, hanging ranking first. **Homicidal cases** are rare, but are sometimes met with in India (see *post*, p. 79).

Way in which death occurs.—The specific gravity of the human body, with the lungs full of air, being less than that of water, an individual falling into water commonly, but not invariably, rises to the surface. Here he begins to struggle, raises a portion of his body out of the water, and, as a consequence, sinks. Presently he again rises to the surface, again begins to struggle, and again sinks. This alternate rising to the surface and sinking below it goes on, the individual each time he rises taking a mixture of air and water into his mouth. Some of the water is swallowed, and some comes into contact with the glottis, causing cough and forcible expiratory efforts. Ultimately, in consequence of the expulsion of air from the lungs, the specific gravity of the body becomes greater than that of water, and the body ceases to rise.

Mode of death.—In 87·5 per cent. of cases, death occurs either by pure apnœa (25 per cent.), or a combination of apnœa with syncope or cerebral congestion (62·5 per cent.). In the remaining 12·5 per cent., death takes place by apoplexy, or

cerebral congestion, or syncope, or shock (asthenia). Death by shock is met with in cases where the individual, in falling into the water, strikes his head, chest, or abdomen, forcibly against some hard object, or against the surface of the water simply, if the fall is from a great height.

Period at which death takes place.—This varies with the mode of death; is instantaneous if from shock, rapid if from pure apnœa, less rapid if from a combination of apnœa with syncope or cerebral congestion. When death occurs from pure apnœa, asphyxia commences as an outside limit after two minutes' complete submersion, and death takes place within five minutes. Recovery is rare after five minutes' complete submersion. Treatment should, however, be persevered with, until it is certain that death has taken place, (*a*) because in exceptional cases animation has been restored after more than five minutes' complete submersion, (*b*) because the submersion, although alleged to have been complete, may not have been so, and (*c*) because by persevering treatment, individuals have been recovered, who have shown no signs of animation for several hours; in one case of recovery it is said that there were no signs of animation for 8½ hours.

Treatment should be commenced at once. First get rid of any water in the mouth, &c., by placing the body for a few seconds face down with the head a little lower than the feet, keeping the mouth open, and the tongue drawn forwards. Next turn the body on the back and commence artificial respiration. As quickly as possible, the body should be stripped, rubbed dry, and warmth applied to the surface. Weak ammonia may be held to the nostrils, and galvanism employed (one pole to the nape of the neck and the other to the epigastrium). All crowding round the patient should be avoided.

Artificial respiration.—There are three principal methods of employing artificial respiration, viz. (1) Marshall Hall's, (2) Silvester's, and (3) Howard's direct method. Of these No. 2 is better than No. 1, and No. 3 is said to be better even than No. 2.

(1) **Marshall Hall's method.**—This consists in turning the patient alternately on his face and side, making presssure on the back each time the patient is turned on his face. When the patient is turned on his face, his chest must be supported on some suitable cushion, and his head should be supported and kept raised by placing one of his arms under his forehead.

(2) **Silvester's method.**—In this the patient is laid on his back with his head and shoulders slightly raised. The operator takes up his position behind the patient's head, grasps the patient's arms just above the elbows, one in either hand, draws them up above the patient's head in a line with the patient's body, and keeps them well stretched upwards for two or three seconds. The patient's arms are then to be turned down and pressed for a

couple of seconds firmly against the sides of the patient's chest, then raised again, and again pressed down about fifteen times a minute.

(3) **Howard's direct method.**—Dr. Howard's instructions for carrying out this method are as follows:—

"(*a*) **Instantly** turn the patient **face downwards** with a large firm roll of clothing under the stomach and chest. Press with your weight two or three times, for four or five seconds each time, upon the patient's back, so that the water is pressed out of the lungs and stomach, and drains feebly downwards out of the mouth. Then (*b*) **Quickly** turn the patient **face upwards**, the roll of clothing being now put **under his back just below the shoulder blades**, the head hanging back as low as possible. Place the patient's hands together above his head; kneel with the patient's hips between your knees; fix your elbows against your hips. Now, grasping the lower part of the patient's chest, squeeze the two sides together, pressing gradually forward with all your weight for about three seconds until your mouth is nearly over the mouth of the patient; then, with a push, **suddenly** jerk yourself backwards. Rest about three seconds, then begin again. Repeat these bellows-blowing movements, so that the air may be sucked into the lungs about eight or ten times a minute. **Remember** the above directions must be used **on the spot**, the instant the patient is taken from the water. A moment's delay and success may be hopeless. As soon as the water is pressed from the lungs, all clothing should be ripped away from the chest and throat. In making the pressure either for the removal of the water or for breathing, increase it **gradually** and thoroughly, and **suddenly** let go with a jerk. With women and children use less force. Do not stop these movements under an hour unless the patient breathes. Be careful not to interrupt first short natural breaths. If they be long apart, carefully continue between them the bellows-blowing movements as before."

Medico-Legal Questions.

The chief medico-legal questions connected with drowning are:—(I.) Was death due to drowning or to some other cause operating before immersion? and (II.) Was the drowning accidental, suicidal, or homicidal?

Question I. Was death due to drowning or to some other cause operating before immersion?

As in 87·5 per cent. of cases of death from drowning, the mode of death is apnœa, pure or mixed, the post-mortem appearances of death from apnœa will usually, but not invariably, be

found. Whether, however, the post-mortem appearances of apnœa are present or not, other appearances indicative of death from drowning must be searched for because (*a*) apnœa, if present, may have been the result of causes other than drowning, and (*b*) the mode of death may not have been apnœa, and yet death may be due to drowning. **The post-mortem appearances other than those of apnœa** which indicate death by drowning are as follows:—

1. **Froth in the mouth, nostrils, and bronchi.**—This, although usually present in death from drowning, may be absent, especially in cases where the body has not risen to the surface after its first submersion. It is often also present in death from causes other than drowning, *e.g.*, in epilepsy, and in cases of death from apnœa not due to drowning.

2. **Water in the stomach,** especially if this contains matters such as are present in the water of immersion, *e.g.*, algæ, diatoms, &c. Water is always found in the stomach, if the individual was sensible at the time of immersion. It is highly improbable that after death, water can enter the stomach, hence the presence of this post-mortem appearance indicates it to be highly probable (*a*) that the individual was sensible at the time of immersion, and (*b*) that as a consequence death was due to drowning. On the other hand, the absence of this post-mortem appearance indicates that the individual was insensible (not necessarily dead) at the time of immersion.

3. **Mud, sand, or floating matters mixed with water in the lungs or windpipe.**—This is evidence of even greater value than the last-mentioned appearance in favour of the supposition that the individual was sensible at the time of immersion, and that, therefore, death was most probably due to drowning.

4. **Sand, mud, weeds, sticks, &c, grasped in the hands or sticking under the nails,** are evidence of struggles in the water during life, and hence presumptive evidence in favour of death having been caused by drowning.

5. **Other minor signs. Cutis anserina,** or goose skin, if present, indicates that immersion took place either during life or shortly after death. No conclusion can, however, be drawn from the absence of this appearance.—**Retraction of the penis.**—This, according to Casper, if present, affords evidence of some weight in favour of drowning. Ogston, however, found this condition present in only six out of thirty cases.

Lastly it should be noted that **the post-mortem appearances of death by drowning may be altogether absent,** and yet drowning may have been the cause of death. Obviously in such cases the absence of appearances indicative of death from a cause other than drowning, *e.g*, violence, poison, or disease, must be ascertained by careful search.

Question II. Was the drowning accidental, suicidal, or homicidal?

Frequently no indication as to this is afforded by the post-mortem appearances. It may be, however, that—

1. **Marks of violence are present on the body.** Very often, however, such marks are due to accidental injury at the time of immersion, or, but less often, to injury after immersion. Hence in a case of death from drowning such marks do not indicate homicide, unless from their nature or from the circumstances of the case, the possibility is excluded of their being due to—(*a*) **Injuries received at the time of immersion,** owing to the body striking in its fall against some hard object, or if the fall has been from a great height against the surface of the water. Bodies found in wells frequently exhibit severe injuries caused in the first of these two ways; and fracture and dislocation of the cervical vertebræ has resulted from the head striking forcibly against the bottom of a shallow bath. Again a case is recorded in which dislocation of both arms backwards was caused, by the body after falling from a great height striking the surface of the water with the arms outstretched. (*b*) **Injuries received after immersion,** during life or after death, *e.g.*, a case is reported where a mark of a ligature on the neck was produced by the string of a cloak getting tightly drawn round the neck during the struggles of the drowning person; and another where fracture of one of the cervical vertebræ was caused by the muscular effort of throwing the head violently back on contact of the body with the water. Obviously also severe injuries may result from the body during life or after death being forcibly dashed against some hard object, *e.g.*, a rock or wall, or the pier of a bridge; or from the bites of animals.

2. **Ligatures** are found round the hands or feet, or weights are found attached to the body. In such cases accident is contra-indicated. If the ligatures are found tied in such a way that the individual himself could not have tied them (but not unless this is the case) suicide also is contra-indicated.

3. **The body is found in shallow water.**—In this case accident is contra-indicated, unless the individual was intoxicated or insensible at the time of immersion. Suicide is not contra-indicated, as cases are known of individuals drowning themselves in water only a few inches deep. If drowning in shallow water is homicidal, marks of violence due to the force employed in holding the victim under water will usually be present. Here it may be mentioned that in some parts of India a form of ordeal, to which women suspected of witchcraft are in some instances subjected, is holding the head under water during the time an arrow is shot from a bow, and brought back to the place from which it was shot.[1]

[1] Gribble, Med. Jurisp. for India, p. 154.

HANGING.

Origin of cases.—Hanging is the judicial mode of inflicting capital punishment in India, England, and many other countries. **Suicidal cases** are common in all countries. In India, statistics (see Table VII, *Appendix*, page 496), show that between one-third and one-half of the suicides of both sexes in the town of Calcutta and in the Punjaub, and of the male suicides in the presidencies of Madras and Bombay, hang themselves. Hanging also is the mode selected by about 18 per cent. of the female suicides in the two last-mentioned presidencies. **Homicidal cases** are rare, but are sometimes met with in India. Chevers mentions three, one where a woman with the aid of three men hung her husband in revenge for his having beaten her some days previously; another where a husband hung his wife as a punishment for adultery; and a third where the inhabitants of a village, discovering a man from a neighbouring village in the act of committing a theft, hung him on a tree in the middle of their village. More frequently in India, in homicidal cases where the body is found hanging, the cause of death is strangulation or mechanical violence, and the body has been hung to avert suspicion (see *post* and *Cases* A 23 and 24). **Accidental cases** also are rare, but are sometimes met with.

The mode of death in hanging depends on the way the cord is applied and on other circumstances. It may be by

1. **Fracture or dislocation of the cervical vertebræ** followed by almost instant death from pressure on the spinal cord. This occurs when the body falls some distance before the strain comes on the rope, and is the mode of death sought to be attained in judicial hanging.

2. **Apnœa**, from constriction of the air passages. Death from pure apnœa does not often occur in hanging. It may however occur if a knot or some hard object contained in the ligature presses directly on the trachea.

3. **Apoplexy** from pressure of the ligature on the large veins of the neck.

4. **Mixed apnœa and apoplexy.**—This, except in judicial hanging, is the most common mode of death, occurring in about 77 per cent. of those cases of death from hanging in which the cause of death is other than fracture of the neck.

The rapidity with which death occurs varies. It occurs almost instantaneously if the neck is fractured, rapidly if death takes place by apnœa; and least rapidly if apoplexy is the mode of death. If there is no injury to the spinal cord, and the stoppage of air is not complete, five to eight minutes is the common fatal period; but it is possible that life may be restored after even half an hour's suspension [1] (see *Case* A 22).

[1] Tidy's Leg. Med. II., p. 391.

Treatment.—As in drowning (see p. 76). In addition, moderate bleeding may be employed to relieve cerebral, or general venous congestion.

Post-mortem Appearances.—These may be classed under three heads, viz.:

1. **Appearances indicative of the "mode" of death,** *e.g.*, fracture or dislocation of the cervical vertebræ; or the post-mortem appearances of apnœa or apoplexy, one or both.

2. **The mark of the cord.**—This in death from hanging is usually but not always oblique and non-continuous, *i.e.*, does not completely encircle the neck. It is usually (in about 81 per cent. of cases) situated between the chin and the larynx, and is very seldom (in about 2 per cent. of cases) below the larynx. In appearance it is usually a well-defined furrow, which, according to the length of the period of suspension, may (*a*) show no change of colour, or at most a red blush, or (*b*) be condensed and white at the bottom, the edges presenting either no change of colour, or being red, and the skin beyond violet, or (*c*) if the period of suspension has been long, be dry, hard, yellowish-brown, and horny, resembling parchment. Abrasions are sometimes found in the course of the mark, but ecchymosis is rare. After noting the exact situation and external appearance of the mark on the neck, two incisions should be made round the neck, one about an inch above, and the other about an inch below the mark. These should be connected at the back of the neck by a vertical cut, and the skin carefully dissected up from behind forward. The subcutaneous cellular tissue will then usually be found to show a condensed white or yellow line. The underlying soft parts and the spine should then be examined. Such dissection should be made also in all cases where strangulation is suspected. Much local injury may be found in cases where, as in judicial hanging, the body falls some distance before the strain comes on the cord. Such cases excepted, much local injury points to strangulation rather than to hanging; or if hanging be the cause of death, to homicidal, rather than to suicidal hanging.

3. **Other appearances.**—**The face** may be found pale, the features placid, and the eyes not unduly prominent. This Harvey considers to be the most usual condition in fresh bodies; or, especially if decomposition has set in, the face may be found swollen and the eyes protruding. **The pupils** are nearly always dilated. **The tongue** is usually found swollen and pressed against the teeth, or partly protruding between them and bitten. **The genital organs** frequently show signs indicative of excitement, accompanied by discharge of mucus, or sometimes of blood, and in males by emission of seminal fluid. Expulsion of urine and fæces sometimes takes place.

The lungs.—Congestion of the lungs is by no means invariably present. Out of 834 cases of death from hanging included in

the returns reported on by Harvey, in 738 the lungs were congested, in seventy-seven natural, and in nineteen collapsed. Rupture of the superficial air cells, spots of sub-pleural ecchymosis, and apoplectic effusions into the substance of the lungs, are all infrequent in hanging, but may be present.

Marks of Saliva running in straight lines down the chin and chest are usually found, and if present are important as indicating suspension during life.

Medico-Legal Questions.

The chief medico-legal questions connected with death by hanging, are:—(I.) Was death due to hanging? and (II.) Was the hanging suicidal, accidental, or homicidal?

Question I. Was death due to hanging?

With reference to this question, it may first be pointed out that in a case of death from hanging, where the period of suspension has been short, or a very soft ligature has been used, there may be no mark at all on the neck. **Hence the absence of a ligature mark on the neck does not absolutely contra-indicate hanging** as the cause of death.

The presence of a ligature mark on the neck does not necessarily indicate suspension of the body (see *Strangulation*). A ligature mark due to suspension of the body is, as a rule, high up on the neck, oblique, and non-continuous. Suspension of the body, therefore, is indicated by the presence of a ligature mark on the neck, with a force proportionate to the degree of agreement of the mark, with these characters. In very exceptional cases, however, a mark possessing all these characters, may be produced without suspension; *e.g.*, when the body has been dragged along the ground, during life or after death, by a ligature round the neck. In such a case, abrasions of the skin, due to the dragging, will probably be found (see *Case* A 15). Suppose, however, **suspension of the body** to be **proved** by direct evidence, or strongly indicated by the characters of the ligature mark on the neck, death may yet have been due to a cause other than hanging, and the suspension of the body effected after death. That a ligature mark on the neck, in all respects resembling the mark left by the cord in a case of death from hanging, may be produced by suspension of the body after death has been amply proved. Casper found that such a mark was produced when bodies were suspended within two hours after death; and Tidy states that an ecchymosed mark may be produced within three, and a non-ecchymosed mark within six hours, after death. This being so, to establish the fact that death was due to hanging, requires not only proof of suspension of the body, but also **proof**

that such suspension was the cause of death. Such proof may be afforded by the presence of the general post-mortem appearances, already enumerated, of death from hanging. If these are absent, careful search should be made for the presence of signs of death from a violent cause other than hanging. This is extremely important, as murder cases are not infrequently met with in India, in which the murderer suspends the body of his victim after death, with the object of imitating suicidal hanging (see *Cases* A 23 and 24). In such cases (see *Cases* A 23) the cause of death sometimes is strangulation, and when this is so, the general post-mortem appearances present may closely resemble those of death from hanging (see *Strangulation*).

Question II. Was the hanging suicidal, accidental, or homicidal?

In cases of death from hanging **the presumption is always greatly in favour of suicide.** This presumption is but little weakened by the body being found only **partly suspended.** Numerous suicidal cases are on record in which the body was found partly suspended, with the feet touching the ground, or in a sitting, reclining, or kneeling posture. Ogston mentions a remarkable case of this kind (see *Case* A 25). Suicide, however, may be negatived by the body being found suspended in such a manner as to show that the individual could not have hung himself. Again, supposing the post-mortem appearances to show that death was due to hanging, the discovery of an **irritant poison**—*e.g.*, arsenic—in the body but little affects the presumption in favour of suicide. Harvey mentions two cases in which individuals, after having taken arsenic, hung themselves apparently in order to escape the suffering caused by the action of the poison. (See, however, *Case* A 27.) Death being due to hanging, marks of **mechanical violence** present on the body only positively contra-indicate suicide when the violence indicated is sufficient to have caused immediate insensibility. Mechanical violence short of this, if from its characters self-inflicted, strengthens the presumption in favour of suicide. That suicidal hanging may follow self-infliction of a very severe wound, is shown by a case cited by Harvey, in which a man hung himself after inflicting a wound on his throat four inches long, dividing the thyroid cartilage and œsophagus. Non-self-inflicted violence, not sufficient to have caused immediate insensibility, may be present in a case of suicide, and in fact form the motive leading to it.

Accidental hanging is rare. It is sometimes however met with, chiefly in cases where children have been playing at hanging. Cases also are recorded where individuals giving a hanging exhibition have been allowed to remain too long suspended, with

fatal results. In one case an adult was found accidentally hung in a gymnasium; and a very exceptional case of accidental hanging is mentioned by Ogston (see *Case* A 26).

Homicidal cases are also rare. A few, however, are on record where hanging pure and simple appears to have been the cause of death; *e.g.*, the three cases mentioned by Chevers, referred to at p. 80. In such cases, as a rule, a number of persons are concerned in the murder. A person, however, who is weak, or insensible, or even asleep, may be murdered by hanging by a single other individual. Ogston, for example, mentions a case "where a woman tied a ligature round the neck of her husband while he was asleep, and then pulled him up." Cases are more common where individuals are first rendered insensible (or, it may be, killed) by mechanical violence, or by strangulation, and then subsequently hung. **The presence of marks of self-inflicted mechanical violence** tends, as already pointed out, to strengthen the presumption of suicidal hanging. When marks are present, clearly due to the infliction of mechanical violence by another, such marks may indicate the employment of violence sufficient to have caused (*a*) death, or (*b*) immediate insensibility, or (*c*) insufficient to have caused either of these effects. In case (*a*), the absence of the general post-mortem appearances of death by hanging obviously confirms the indication of homicide. In case (*b*), the general post-mortem appearances of death by hanging may be present, but still homicide is indicated. In case (*c*) it is often quite impossible from the post-mortem appearances, to arrive at any conclusion as to whether the hanging was suicidal or homicidal. **If strangulation has been employed** previous to suspension, evidence of this may be afforded by the presence on the neck, in addition to the mark due to suspension of the body, of marks indicating strangulation (see *Strangulation*). It may here, however, be pointed out that two cord marks on the neck, one having the characters of a strangulation, and the other those of a hanging mark, may be found in a case of simple hanging, if the cord has been passed twice round the neck. If very **severe injuries** are found to have been **produced by the cord**, *e.g.*, laceration of the muscles or other underlying soft parts, the presumption is generally in favour of homicide. Much injury to the soft parts may, however, be met with in suicidal hanging, if the individual has arranged matters so that his body falls some distance before the strain comes on the cord. Homicide is obviously indicated if the position in which the body is found suspended, or the presence of ligatures securing the hands, show that the individual could not have hung himself.

STRANGULATION.

Strangulation includes all cases of death from external pressure applied to the neck by means other than a ligature kept tight by the weight of the body. Unlike hanging, strangulation may be effected without a ligature, *e.g.*, by pressure with the fingers, or some hard object. The modes of death in strangulation are the same as in hanging, hence the post-mortem appearances are also very similar. The main points of difference between the post-mortem appearances of strangulation and those of hanging, are as follows:—

1. **The mark or marks on the neck.—If a ligature has been used,** there will, save in very exceptional cases, be found a mark on the neck. This usually, but not invariably, differs from a hanging mark, in being transverse in direction, low down on the neck, and continuous, *i.e.*, completely encircling the neck. In exceptional cases of strangulation, especially if the body has been dragged by the ligature, the mark may be found high up on the neck, and oblique in direction, like a hanging mark. Again, in exceptional cases of hanging, the mark may be found low down on the neck, and, if the cord has been tightly applied, the mark left by it may be transverse in direction, like a strangulation mark. Abrasions and ecchymoses in the course of the mark, and injury to the underlying soft parts, are much more common in strangulation than in hanging; but the hard yellow brown parchmenty appearance of the skin in the course of the mark is more seldom met with. **Strangulation by manual pressure** is tolerably common in India, the victim being usually a child or a female. Where this mode has been employed, marks of violence are invariably, or almost invariably, found on the neck, sometimes, however, only visible on dissection. Usually the marks found on the neck in such cases clearly indicate how the strangulation has been effected. **Strangulation by compression of the neck with a stick or other hard substance,** although not so common as strangulation by hand, is still often met with in India. Usually, one stick placed across the front of the neck is used; but sometimes two sticks are employed, one placed behind, and the other in front of the neck. This mode of strangulation causes a central bruise on the front of the neck, and usually, severe local injury.

2. **The appearances presented by the thoracic viscera.—The lungs,** as in hanging, may be found uncongested. According to Tardieu, patches of emphysema on the surface of the lungs, due to rupture of the superficial air-cells, are invariably, and apoplectic effusions into the substance of the lungs commonly, present in death from strangulation; while punctiform sub-pleural ecchymoses (Tardieu's spots), common in suffocation, are rare in strangulation. These appearances are all infrequent

in hanging. **Effusion of bloody serum into the pericardium** is very common in death from strangulation, and comparatively rare in death from hanging; it, however, is present in some cases of hanging, especially when death has occurred slowly.

3. **Marks of saliva** running in straight lines down the chin and chest, a common appearance in death from hanging, are not likely to be present in strangulation.

Medico-Legal Questions.

As in hanging, the chief medico-legal questions connected with death by strangulation, are:—(I.) Was death due to strangulation? and (II.) Was the strangulation homicidal, suicidal, or accidental?

Question I. Was death due to strangulation?

It may first be pointed out, that in very exceptional cases, death may occur by strangulation, without any mark being present on the neck. This may happen if a soft ligature has been used. It rarely, however, occurs, as even when a soft ligature is employed, much superfluous violence is commonly applied, and a distinct mark on the neck is usually present. If no marks of violence, either external or internal, are to be found on the neck, strangulation is very strongly, but not positively, contra-indicated. **When strangulation has been effected by means other than the use of a ligature** much violence is almost always used —often to other parts of the body as well as the neck—and there is seldom any difficulty in arriving at a conclusion as to the cause of death. Here, however, it must be remarked, that in very exceptional cases of death from natural causes, finger marks may be found on the neck, accompanied by the post-mortem appearances of death from apnœa. An individual dying from apnœa the result of disease, *e.g.* epilepsy, may, in his struggles for breath, by clutching at his throat, produce such marks (see *Case* A 28). Hence, if the only marks of violence present on the body are slight finger marks on the neck, a guarded opinion must be given as to the cause of death.

A ligature mark present on the neck corresponding in external appearance to a strangulation mark, cannot of itself be taken as evidence of death by strangulation. Such a mark may be the result of the application of a ligature to the neck after death, or have been accidentally produced by the pressure of a tight-fitting article of dress, or be the result of putrefactive swelling against a string tied loosely round the neck. In the last two cases, however, injury to the underlying soft parts, common in strangulation, is not likely to be found. Hence, even when a ligature mark is found on the neck, corresponding in

appearance to a strangulation mark, to establish the fact that death was due to strangulation, requires proof that the pressure of such ligature was the cause of death. Such proof may be afforded by the presence of the general post-mortem appearances of death by strangulation. It must, however, be recollected that in hanging, as well as in strangulation by a ligature, death is due to the pressure of a ligature on the neck. Further, that in hanging, the presumption is always in favour of suicide, while in strangulation, it is in favour of homicide. Hence, in all cases of death from pressure of a ligature on the neck, all appearances indicating the cause of death to be hanging, rather than strangulation, or *vice versâ*, should be most carefully noted (see p. 85).

Question II. Was the strangulation homicidal, suicidal, or accidental?

Accidental cases are rare, a few, however, are on record (see *Cases* A 29, 30, and 31).

Suicidal cases of strangulation by a ligature are sometimes met with. To effect suicide in this way requires the employment of some means whereby the ligature is kept tight, independently of any muscular effort on the part of the suicide; so that relaxation may not occur when insensibility supervenes. This end may be arrived at in various ways, *e.g.* by simply passing the ligature more than once round the neck; or, by securing the ends of the ligature to the foot, or to the wrists, in such a manner that the ligature is tightened when the arms are extended; or to some fixed object. Or, it may be arrived at by twisting a stick in the tied ligature, and securing the end of the stick; or by simply knotting the ligature. As regards this last method, it may be noted that the presence of more than one knot raises a suspicion of homicide, two knots have, however, occasionally been found in suicidal cases; more than two knots very strongly indicates homicide.

Homicidal cases.—Just as in hanging, the presumption is always in favour of suicide, so in strangulation the presumption is always in favour of homicide. Homicide is very strongly indicated, (*a*) when a ligature has been employed, by the absence of evidence indicating the use of some means for the purpose of keeping it tight after insensibility has occurred; (*b*) by the presence of signs indicating the application of much violence to the neck or to other parts of the body, and (*c*) when the strangulation has been effected by means other than the use of a ligature. Obviously homicide also is almost conclusively indicated, if the hands are found tied together in such a way as to show that they could not have been so secured by the individual himself.

SUFFOCATION.

Includes all cases of Apnœa (drowning excepted) caused by violent means other than direct pressure on the wind-pipe, *e.g.*:—1. By closing the mouth and nostrils; 2. By pressure on the chest; 3. By closure of the glottis.

1. **Closing the mouth and nostrils.**—This may be (*a*) **Homicidal,** as in cases of infanticide effected by closing the mouth and nostrils with the hand. The mouth and nostrils in homicidal cases, may also be closed by plasters applied to the face; this was the way in which the resurrection men, Burke and Hare, murdered their victims in Edinburgh. Burke, after conviction, confessed to sixteen murders effected in this way in a few months. Again, soft pillows may be employed, as in the case of the two Princes murdered in the Tower of London. (*b*) **Accidental,** as in cases where children are accidentally suffocated by their mothers overlying them in bed. (*c*) **Suicidal.** Cases of suicide effected in this way are extremely rare. Taylor, however, mentions a case of a woman who is reported to have committed suicide, by simply leaning with her mouth and nostrils pressed against the bed-clothes.

2. **Pressure on the chest.**—Suffocation caused in this way is **generally accidental,** usually occurring from either accidental burial under the *débris* of falling buildings, earth, &c.; or pressure in a crowd, as in the case which occurred in Paris in 1837, in which twenty-three persons were suffocated in this way, in a crowd in one of the streets. A case also is recorded of a man who, while a plaster cast of his trunk was being taken, was nearly killed by the pressure on his chest of the solidifying plaster. **Homicidal cases** are sometimes met with in India. In homicidal cases, if the victim is an adult, and was not first rendered insensible, or was not a consenting party, probably several persons will be found to have been concerned in the murder. Often great violence is used, sometimes causing symmetrical, or nearly symmetrical, fractures of the ribs (see Wounds, p. 63). In children, great violence may be employed, sufficient, in fact, to cause extensive injury to the lungs, without fracture of the ribs (see *Case* A 37 *b*). If the pressure on the chest has been steady and long continued, it is probable, as pointed out by McLeod, that, broad horny marks will be found on the skin. Under the head of homicidal suffocation by pressure on the chest, may also be mentioned (1) the burial alive of widows with their husbands' body, a custom formerly prevailing, to a certain extent, in India, and (2) the "Samádh," or burial alive of lepers—often with the consent or at the entreaty of the victim—cases of which used formerly to be not

infrequently met with in India.[1] **Suicidal suffocation** by pressure on the chest is hardly possible.

3. **Closure of the glottis.**—Suffocation thus caused often occurs **accidentally** from the impaction of foreign bodies—masses of food, for example—in the throat; or from spasm of the glottis, the result of disease or of the inhalation of poisonous gases. **Suicidal suffocation** by closure of the glottis, effected by forcing rags, articles of dress, &c., into the fauces is sometimes met with. **Homicidal cases** are rare in adults. Children in India, however, are sometimes murdered by filling their mouths with mud or other soft material (see *Cases* A 35, 36), this also is a common method of infanticide in India.

The post-mortem appearances of death from suffocation by violence are:—

1. **The post-mortem appearances of apnœa.**—Cases, however, have occurred of undoubted death from suffocation, where most of the post-mortem appearances of apnœa were absent. On this point, Christison, in the case of the woman Campbell, murdered by Burke, the resurrectionist, remarked, "the conviction in the public mind, that a well-informed medical man should always be able to detect death by suffocation, simply by an inspection of the body, and without a knowledge of collateral circumstances, is erroneous, and may have the pernicious tendency of throwing inspectors off their guard, by leading them to expect strongly marked appearances in every case of death from suffocation. That such appearances are very far from being always present, ought to be distinctly understood by every medical man."

2. **Punctiform sub-pleural ecchymoses** (Tardieu's spots), usually round, well-defined, dark coloured, and varying in size from a pin's head to a small bean, are frequently, but not invariably, found on the surface of the lungs. Their usual position is at the root, base, and lower margin of the lungs. They may also be found in other situations, *e.g.*, under the scalp, on the pericardium, and especially in infants, on the thymus gland. Tardieu's spots, however, may, especially in adults, be absent in cases of suffocation, and present in cases of death from causes other than suffocation (see *post*). Apoplectic effusions into the substance of the lungs, common in strangulation, are not met with in cases of suffocation (Tidy).

3. **Appearances indicative of violence such as would cause suffocation.**—*e.g.*, marks of violence on the chest, marks indicating the application of manual pressure, or of plasters over the mouth and nostrils, foreign bodies impacted in the throat, &c.

[1] Cases of live burial of lepers occurred in Rajputana in 1868. Harvey (Beng. Med. Leg. Rept., 1870–72) points out that the victim is often buried in a squatting posture up to the neck only, with the earth loosely packed around him, death taking place from exposure and exhaustion. After death the head is covered with earth.

Cases, however, of death from suffocation by violence may occur, and no appearance of this class be present.

Medico-Legal Questions.

As in hanging and strangulation these are:—(I.) Was death due to suffocation? and (II.) Was the suffocation accidental, suicidal or homicidal?

Question I. Was death due to suffocation?

The chief points bearing on this question are:—

1. **The post-mortem appearances of apnœa may be nearly entirely absent,** and yet death may have been caused by suffocation (see Christison's remarks just quoted).

2. **The post-mortem appearances of apnœa may be present, and those of drowning, hanging, and strangulation absent,** and yet death may not have been the result of suffocation by violence, but may have been due to apnœa, the result of disease, or poison, *e.g.*, epilepsy, tetanus, or strychnia poisoning. Hence, in cases of alleged suffocation by violence, much depends on the presence or absence of **signs indicative of the employment of violence,** such as would produce suffocation. If these are absent, no positive opinion can be given, from the post-mortem examination alone, as to the cause of death.

3. **Tardieu's spots,** if numerous, well-defined in shape, and limited in size, present on the lungs and thymus gland, strongly contra-indicate strangulation, and **indicate suffocation** to be the cause of death. **Their presence,** however, is **consistent with death from causes other than suffocation.** They have been met with in the bodies of adults after death from drowning, hanging, strangulation, scarlatina, heart disease, apoplexy, pneumonia, &c.; and in the bodies of still-born, and even unborn infants. Further, **their absence does not,** at any rate in the case of adults, conclusively **contra-indicate suffocation.** Ogston failed to find them in nine cases of death from suffocation in adults.

Question II. Was the suffocation accidental, suicidal, or homicidal?

1. **If the deceased is an adult,** the presumption is always in favour of accident. Curious accidents leading to suffocation by closure of the glottis sometimes occur (see *Cases* A 32, 33 and 34). Suicidal cases are rare, but are sometimes met with, *e.g.*, the case of suicide by closure of the mouth and nostrils already referred to. A case also is reported in which a prisoner committed suicide by stuffing his mouth with rags, another in

which a young woman suffocated herself by stuffing a large ball of hay into her throat, and another of a young woman who committed suicide by shutting herself up in a trunk. Homicidal cases are not often met with. In a homicidal case, unless the victim was suffocated while insensible, marks showing the employment of much violence will probably be found.

2. **If the deceased is a child or infant,** suicide is of course contra-indicated, and the question lies between accident and homicide. Accidental cases often arise from overlying, or from accident during birth (see *Infanticide*). Homicidal cases are sometimes met with in children (see *Cases* A 35 to 37), and are frequently met with in infants; stuffing the mouth with rags, or filling it with cow-dung or dirt, being common modes of infanticide in India (see *Infanticide*).

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CHAPTER V.

BURNS AND SPONTANEOUS COMBUSTION.

BURNS.

BURNS are injuries caused by the application of heated substances to the body. If the heated substance is a fluid, the injury is commonly termed a scald. Injuries caused by the application to the surface of the body of corrosive substances, *i.e.*, such substances as cause chemical destruction of the tissues, may also, for medico-legal purposes, be classed as burns.

The chief medico-legal questions connected with burns are :—(I.) Is the injury a burn, and if so, how was it caused? (II.) Was the injury inflicted during life? (III.) Was the injury the result of accident, self-inflicted, or inflicted by another? and (IV.) What results followed, or are likely to follow, from the injury?

Question I.—Is the injury a burn, and if so, how was it caused?

A conclusion arrived at in regard to this question may be of importance, because—

1. By sects. 324 and 326 of the Indian Penal Code, the causing of hurt, or grievous hurt, by certain specified means, is made an offence punishable more severely than when such means have not been used. Among the means specified in these two sections are not only what may be called lethal weapons (see *Wounds*, p. 40), but also "fire or any heated substance," or any "corrosive substance," or "explosive substance."

2. It may affect the question of the guilt or innocence of an accused person. In this respect it may be of much importance to determine, if possible, the precise means whereby the injury, if a burn, was produced, *e.g*, whether produced by the application of a particular heated solid, or of a heated liquid, or by the application of a particular corrosive substance.

For medico-legal purposes, injuries caused by the application of heated substances to the body may be divided into: 1. Burns producing mere redness. 2. Burns causing mere vesication. 3. Burns causing the death of the part injured. And to these

three classes may be added a fourth, viz.: 4. Burns caused by the external application of corrosive substances.

1. **Burns producing mere redness** are usually caused by the momentary application to the part of a hot solid, or of a fluid at a temperature several degrees below the boiling-point of water. They are followed by superficial inflammation, with or without desquamation of the cuticle. Burns of this class may be simulated by the application of various mild irritants to the skin.

2. **Burns causing vesication** are produced by the application of liquids at a temperature about that of boiling water; or by the momentary application to the part of a flame, or of a highly-heated solid. Burns of this second class, caused by flame or by highly-heated solids, may be accompanied by blackening of the skin and scorching of the hair at the seat of injury. In mild burns of this second class, the vesicles simply dry up and heal, and no permanent marks are left. In severe cases, or in unhealthy subjects, suppuration of the vesicles may occur, followed by ulcers, leaving permanent cicatrices. Burns of this second class may be simulated by the application to the skin of various strong irritants, *e.g.*, cantharides and tartar emetic. In badly-nourished persons vesication of the skin, resembling a burn, may occur without the application of heat.[1]

3. **Burns causing the death of the part injured** are produced by prolonged contact with flame or with highly-heated solids, or by contact with liquids at a temperature considerably above that of boiling water, *e.g.*, boiling oils or melted metals. They vary in appearance and degree of gravity, according to the depth to which the injury extends, *e.g.*, the death of a portion of the skin only may have been caused (*Dupuytren's 3rd and 4th degrees*); or the underlying soft parts, as well as the skin, may be affected (*Dupuytren's 5th degree*); or an entire limb, bones and all, may be destroyed (*Dupuytren's 6th degree*). Burns of this class often leave sores difficult to heal, or so large in extent as to require the performance of an amputation. They leave permanent cicatrices, which frequently contract considerably, causing by their contraction considerable deformity, or impairment of the use of members or joints. If a burn of this class has been caused by the application of a heated solid, the form of the solid employed may frequently be inferred from the shape of the burn.

It may be noted, in regard to burns produced by highly-heated solids or liquids, that the skin, if moist, may be brought into contact for a short time with substances at a high temperature, *e.g.*, red-hot solids or melted metals, without a burn being produced. This depends on the assumption of the spheroidal state by the moisture on the surface of the skin, and as a high tem-

[1] Guy's 'Forensic Medicine,' p. 337.

perature is necessary for the production of this spheroidal state, the temperature of the substance brought into contact with the skin must be high, otherwise a burn will be produced.

4. **Burns caused by the application of corrosive substances to the body** seldom extend deeper than the true skin. Vesication does not accompany burns of this description, and there is no scorching of the hair in the neighbourhood of the burn. Further, if the corrosive substance is, as is commonly the case, a liquid, marks left by it will usually be found on the clothes of the person injured. The particular corrosive employed may frequently be inferred from the colour of the marks on the skin, or definitely ascertained by chemical examination of the stained portions of clothing (see *Corrosive Poisons*).

Question II.—Was the injury inflicted during life?

This question sometimes arises, *e.g.*, in cases where, in order to conceal a murder, an attempt is made to burn the body of the murdered person. The chief appearances whereby burns inflicted during life may be distinguished from post-mortem burns are the presence of (1) signs of inflammation; (2) A line of redness; and (3) vesication.

1. **Signs of inflammation** and reparative action, such as the presence of granulations or pus on the injured surface, indicate that the injury was inflicted some considerable time before death. The absence of such signs, of course, does not indicate that the injury was inflicted after death.

2. **A line of redness.**—If a burn is inflicted during life, in the great majority of cases, a line of redness almost immediately forms round the injured surface. This line of redness, although it may be surrounded externally by a blush, disappearing on pressure or after death, does not itself disappear on pressure, and remains visible after death has taken place. The presence of a line of redness possessing the above characters is almost certain evidence that the burn was inflicted during life, and conclusive evidence that it was inflicted during life, or within ten minutes after death. Its absence, however, is not positive evidence that the burn was inflicted after death.

3. **Vesication.**—Here it is convenient to distinguish between what may be called respectively true and false vesication. In true vesication the vesicles contain serum very rich in albumen. In false vesication the vesicles either contain air only, or (especially in dropsical bodies) a small quantity of serum, in which traces only of albumen are present. The presence of true vesication, as the result of a burn, is proof that the injury was inflicted during life. The presence of false vesication, as the result of a burn, shows that the injury was inflicted after death.

The entire absence of all vesication is quite consistent with the supposition that the burn was inflicted during life.

Question III.—Was the injury the result of accident, self-inflicted, or inflicted by another?

Accidental cases are **common**, so common, in fact, that in temperate climates (and in tropical climates also, but to a less degree) the presumption is always in favour of accident. Accidental cases arise in various ways: an individual's clothes catch fire, or a heated liquid, such as boiling water, is spilt accidentally over him; or a petroleum lamp breaks, and the oil it contains catches fire and falls on him. In a few cases, again, individuals in a state of intoxication or insensibility fall asleep near a fire, and are accidentally burnt to death (see also cases of so-called spontaneous combustion, *post*, p. 100). In the majority of accidental cases, examination of the body throws little or no light on the question whether the injury was or was not the result of accident. It may, however, be noted that—

1. **If there are burns on several distinct and separate portions of the body**, or if a dead body show signs of having been ignited in two or more distinct places, accident is contra-indicated.

2 **The discovery of the body**—in a case of death from burning—**at the spot where ignition first took place**, is quite consistent with the supposition of accident, if the individual was narcotised or insensible at the time ignition occurred.

3. **Marks of violence** present on the body do not necessarily contra-indicate accident. Such marks may, for example, be due to injuries received prior to, or at the time of, accidental ignition. It must be borne in mind, also, that sometimes marks closely resembling wounds are produced as the result of a burn (see *Case* A 40).

Suicidal cases are so **rare** that, in a case of death from burns, the presumption is always strongly against suicide. Taylor, however, mentions a case where a prisoner attempted to commit suicide by setting fire to his bed, and a case has recently been reported in England of a lunatic, a gardener, who having dug a sort of grave, placed therein a quantity of dry wood saturated with benzoline. He then laid himself, face down, on the wood and set fire to it. The body was almost entirely consumed. **Burns are sometimes self-inflicted** in order to support a **false charge**. Where this is suspected, the question whether or no the injuries correspond in appearance to the alleged method of production must be carefully considered (see *Case* A 38). It must be recollected that the application of the actual cautery, or of moxas, or of strong blistering agents, to the body, is a

favourite method of treatment among *Hakims* in India, and that false charges may be founded on burns so produced. The presence also of such burns on a dead body may give rise to an erroneous suspicion as to the cause of death.

Homicidal cases, and cases of the infliction of hurt by burning, are not infrequent in India. Chevers mentions a number of cases, the means employed being, in many of them, the application of heated iron instruments, *e.g.*, sickles or ladles or spoons, to the part. In other cases, placing the victim over a fire, applying a lighted torch or a piece of ignited charcoal or a heated pipe-bowl, or pouring heated oil on the body, or covering a portion of the body with tow or rags steeped in oil and setting fire thereto, were the means resorted to. In several of Chevers' cases the victims were females, and the burns were inflicted on the pudenda, as a punishment for suspected adultery. In others the victims were children, the burns being inflicted as a punishment for trifling offences. Chevers also mentions numerous cases of the use by dacoits of torture by fire, for the purpose of extorting information from their victims, as to the place of concealment of money or valuables; and also cases in which thieves or persons suspected of theft, have been tortured by burning, as a punishment, or in order to extort confession. Again, plunging the arm into boiling oil, is a form of ordeal to which women suspected of witchcraft are subjected to in some parts of India.

Along with homicidal cases may be classed cases where an attempt is made to **conceal a murder by burning the body** of the murdered person. In such cases nothing but fragments of partially charred bones may be discovered (see *Case* A 39). In other cases the soft parts may be more or less entire, and then two questions obviously arise, viz., (*a'*) Have the burns the character of post-mortem or ante-mortem burns? and (*b'*) Does examination of the body reveal a cause for death (or for the occurrence of insensibility) irrespective of the burns? The first of these questions has already been discussed (see *Quest. II.*). In regard to the second question, the only special point to be noted is, that, as already mentioned, injuries resembling to a certain extent wounds caused by mechanical violence may be produced on a body by the action of heat alone (see *Case* A 40).

Question IV.—What results followed, or are likely to follow, from the injury?

For medico-legal purposes in India this question, as in the case of wounds (see p. 55) becomes: Has the injury caused, or is it likely to cause, death; and if not, has it caused one or other of those forms of hurt which are by the law of India designated as "grievous hurt"? In regard to this question, the following points may be noted.

1. **Death may occur from burns** (1) **Before reaction** sets in, *i.e.*, within about forty-eight hours of the receipt of the injury, from (*a'*) **Shock or collapse**; or, (*b'*) **Coma**, due to congestion of the brain and serous effusion into the ventricles (this may be mistaken for opium poisoning). (2) **After reaction** has set in, from (*c'*) **Various internal inflammations**, *e.g.*, pneumonia, bronchitis, pleurisy, enteritis. Enteritis with ulceration, followed by peritonitis, is a not infrequent cause of death from burning, especially in young people; (*d'*) **Surgical complications** connected with the injury, *e.g.*, gangrene, erysipelas, tetanus, pyæmia, &c.; or (*e'*) **From exhaustion.**

2. **The danger to life in burns** depends chiefly on (*a'*) **The extent of surface injured.**—Burns involving a great extent of surface are specially dangerous to life. "A burn involving two-thirds, or even one-half of the entire skin, may be regarded as certain to destroy life, and the same practically may be said of a burn (if severe) involving one-third of the body" (Tidy). Deep burns involving a limited portion of the body, are not nearly so dangerous to life as burns involving a wide extent of surface. (*b'*) **The part burnt.**—Burns on the trunk are more dangerous to life than burns on the extremities. Death before reaction has set in is specially likely to occur in the case of burns involving a wide extent of surface on the trunk. (*c'*) **The age of the patient.**—Children, as a rule, bear burns badly, whilst old people bear them comparatively well (Tidy).

The first week after a burn is the most fatal period. Erichsen found that in 54 per cent. of fatal cases death occurred within four days, and in 66 per cent. within eight days, after receipt of the injury.

3. **The post-mortem appearances of death from burns.** The soft parts (as in *Case* A 39) may be entirely destroyed, and it may be impossible from the post-mortem appearances to form any opinion as to whether death was due to burning, or to some other cause operating before cremation of the body. If the soft parts are more or less entire, the post-mortem appearances present may be—

(1). **External**: viz., marks on the surface of the body, having the characters possessed by burns inflicted during life, and varying in appearance according to the length of time which has elapsed between receipt of the injury and death.

(2). **Internal.**—In cases of death from burning, no special internal post-mortem appearances may be present; in other cases there may be found:—(*a'*) **Serous effusion** into the cavity of the arachnoid, pleura, pericardium, or peritoneum; (*b'*) **Congestion of the mucous membrane** of the air-passages and of the mucous membrane of the alimentary canal, especially of the œsophagus, duodenum, and ileum (Tidy). Perforating ulcers of the duodenum, resulting from inflammation of Brunner's

glands, are common in cases of death from burns, especially in young children (Curling). Peyer's patches, and the solitary glands generally, are often greatly inflamed and sometimes ulcerated (Tidy); (*c'*) **Congestion of various organs**, *e.g.*, the brain, lungs, liver, kidneys, &c.

In making a post-mortem examination in a case of alleged death from burns it must always be borne in mind that death (or insensibility) may have been produced by causes operating previously to the infliction of the burns. Hence, in cases of alleged death from burning, it is extremely important to note:—(*a'*) Whether or no the burns possess the characters of burns inflicted during life; and (*b'*) Whether the examination of the body reveals any cause for the occurrence of death (or insensibility) other than burning; and if wounds are present on the body, to note whether they appear (as in *Case* A 40) to have been caused by the action of fire or not.

4. **"Grievous hurt" may be caused by burns.**—The injuries which, by sect. 320 of the Indian Penal Code, are designated as "grievous hurt," have already been enumerated. Burns are especially likely to cause the following forms of "grievous hurt":—(*a'*) Hurt **"which endangers life, or which causes the sufferer to be, during the space of twenty days, in severe bodily pain or unable to follow his ordinary pursuits."** It has before been noted that burns involving a wide extent of surface (especially of the trunk) are specially dangerous to life. (*b'*) If the burns are on the head or face, especially if the true skin is affected to any depth, **"permanent disfiguration of the head or face"** is likely to result. (*c'*) **"Permanent privation of the sight of either eye"** is a not unfrequent result of the throwing of corrosive fluids, *e.g.*, oil of vitriol, over the body. (*d'*) **"Permanent impairment of the powers of" a "member or joint"** is specially likely to occur (from contraction of cicatrices) in the case of severe burns in the neighbourhood of joints.

SPONTANEOUS COMBUSTION.

In order to start the combustion of an inflammable substance a portion of it—no matter how small—must be raised to a particular temperature. The temperature required varies with the substance. A mass of phosphorus will take fire if any portion of it be raised to the comparatively low temperature of 140° Fahr. Hydrogen, on the other hand, requires a high temperature for its ignition. Where a portion of an inflammable substance, or mixture of inflammable substances, is capable of acquiring the temperature necessary for ignition either *per se*, or on contact with air only; such substance, or mixture of substances, is liable to catch fire spontaneously, *i.e.*, is liable to spontaneous combustion.

In such substances, the self-acquirement of the temperature necessary for ignition, is the result of the development of heat by chemical action, such chemical action taking place either between the substance and the oxygen of the air, or, in a few cases, between two of the constituents of a mixture. The principal substances liable to spontaneous combustion are—

1. **Certain simple bodies.**—Phosphorus is the best known example of this class. This substance, in its ordinary condition, oxidises in air even at a temperature of 50° Fahr., and requires only a comparatively low temperature for ignition; hence, it is peculiarly liable to catch fire spontaneously. Certain of the metals, if in a finely-divided condition, *e.g.*, iron, are liable to take fire on exposure to air, owing to heat developed by the combination of the metal with oxygen.

2. **Certain compound bodies** take fire at once on exposure to air, *e.g.*, silicon hydride, liquid phosphide of hydrogen, and zinc ethyl. The presence of a small quantity of the vapour of liquid phosphide of hydrogen also, it may be remarked, confers the property of spontaneous inflammability on combustible gases.

3. **Certain mixtures of substances** are liable to spontaneous combustion from:—(*a*′) **The occurrence of chemical action between the constituents of the mixture,** *e.g.*, phosphorus takes fire on being brought into contact with iodine. Many finely-divided metals, and paper moistened with turpentine, take fire in chlorine. Turpentine takes fire if mixed with fuming nitric acid, &c. Probably the occasional spontaneous combustion of red fire (a mixture of sulphur, carbon, antimony sulphide, potassic chlorate, and strontium nitrate) is due to this cause. (*b*′) **The oxidation of one of the constituents of the mixture.**—The constituent undergoing oxidation may be an **inorganic** substance, *e.g.*, a metallic sulphide. Some varieties of coal contain iron pyrites (sulphide of iron) in considerable quantity, and are liable to spontaneous combustion from heat developed by the combination of this with the oxygen of the air. Again, the constituent undergoing oxidation may be an **organic** substance, *e.g.*, a drying oil. Numerous cases are on record of the spontaneous ignition of fibrous and other combustible substances moistened with a drying oil, *i.e.*, an oil capable of drying readily into a resin by taking up oxygen from the air. Cotton, wool, hemp, flax, jute, woody fibre, and lampblack, have all been known to catch fire spontaneously when moistened with linseed or other drying oils. Woody fibre moistened with turpentine, has been known to catch fire from a similar action.

Organic matters moistened with water only, *e.g.*, damp hay, cotton, tow, flax, cocoanut fibre, leaves, &c., are liable to become heated from oxidation; and spontaneous ignition of

damp hay and cotton; and, in one case each, of damp oats and esparto grass, has been known to occur. It may also be remarked that certain **explosive substances** are liable to explode either spontaneously, *e.g.*, chloride of nitrogen; or from a very slight amount of percussion or friction, *e.g.*, nitro-glycerine, the metallic fulminates, and mixtures of combustible substances with potassic chlorate.

The question whether the human body is liable to spontaneous combustion has arisen in the following way: It is well known that in ordinary circumstances, long exposure to a high temperature, and the expenditure of a considerable amount of fuel, is required in order to cause any considerable amount of charring of a human body. Several cases, however, are on record where the bodies of persons, generally old obese females addicted to spirits, have been found near a fire or partly-burned candle, half consumed, and exhaling a fœtid empyreumatic odour. In many of these cases, articles near the body have been found covered with a greasy stinking soot, but although combustible, unburnt, indicating that the temperature of combustion has been comparatively low (see *Case* A 41). Taking into consideration what we know as to the quantity of heat required to effect even partial combustion of a human body, these cases are obviously exceptional, and indicate that, under certain circumstances, the human body acquires a condition of preternatural combustibility; a condition, in fact, in which the body instead of requiring, in order to effect its combustion, a long-continued application of flame; appears to require—for at any rate partial combustion—contact with flame for a short period only. A consideration of the circumstances recorded in authentic cases of this class fails to support the hypothesis of spontaneous combustion, *i.e.*, of ignition without contact with flame, which was at one time put forward to account for them. It may be mentioned that spontaneous combustion has been put forward as a defence in a case of murder, as in the case of the murder of the Countess of Goerlitz (*Case* A 42).

CHAPTER VI.

DEATH FROM HEAT, COLD, ELECTRIC DISCHARGE, AND STARVATION.

DEATH FROM HEAT.

Sunstroke, Insolation, Coup de Soleil, Heat Apoplexy.

The temperature of the human body in health, taken in the mouth or axilla, averages 98°·6 F. In the rectum or vagina it is usually about 1° higher. In many diseases the temperature of the body rises considerably: thus a temperature of 108° F. is common in severe agues; 113° F. has been recorded in typhoid fever, and 115° F. in a case of scarlet fever. Death is inevitable if the temperature of the body rises and remains for any length of time 12° F. above the normal.

Death from heat frequently **occurs without direct exposure** to the sun. According to Dr. H. C. Wood,[1] it may occur in three ways: viz., 1. From exhaustion or collapse. 2. From thermic fever; and, 3, in rare cases, from meningitis or phrenitis. **The principal circumstances which modify the effect** on the system of exposure to heat are:—

1. **The amount of moisture** present in the atmosphere. Other things being equal, the less this is, the better exposure to heat is borne. The presence of a large amount of moisture in the atmosphere interferes with evaporation from the surface of the body, and favours the action of heat on the system.

2. **Length of exposure.**—Very high temperatures can be borne for a short time, but not for long, without ill effects. Chabert, "the Fire King," was in the habit of entering an oven, the temperature of which was from 400° to 600° F.

3. **Habit.**—This appears, to a certain extent, to lessen the effect of exposure to heat. Individuals accustomed to carry on their daily work in an atmosphere of high temperature, apparently withstand the action of heat better than others.

4. **Bodily condition of the individual.**—The action of heat on the system is favoured by exhaustion, indulgence in alcoholic liquors, or anything which checks elimination, or embarrasses the normal working of the organic system. (Tidy.)

[1] Art. in Pepper's System of Med., vol. v., pp. 387, *et seq.*

The symptoms vary.—They may set in suddenly, or there may be premonitory symptoms, such as headache and vomiting. These are followed usually by confusion of vision, flushing of the face, and stupor or coma. The temperature is invariably high. The pupils are generally dilated in the earlier, and contracted to a fine point in the later stages. In a few cases delirium and convulsions are present. Death has been known to occur in five minutes, or as late as three days, after the commencement of the attack.

The post-mortem appearances.—In some cases no abnormal appearance has been present. In the majority of cases, congestion of the brain and its membranes, engorgement of the right side of the heart, and congestion of the lungs and abdominal viscera are found. The blood is frequently fluid and dark in colour.

Death from heat seldom becomes the subject of a medico-legal inquiry.—Taylor,[1] however, mentions a case where the captain of a steam-ship was charged with causing the death of a man, by having him tied up within a short distance of the stoke-hole of a furnace. The man died in a few hours, apparently from the effects of heat.

DEATH FROM COLD.

If, from exposure to cold, the temperature of the human body becomes reduced for any length of time much below the normal, death occurs. In exceptional cases the temperature of the body has been known to fall as low as 79°, or even 75° F., without life being extinguished.

The constitutional symptoms produced by exposure to cold are, depression of the heart's action, and torpor, succeeded by stupor, or coma, from congestion of the nervous centres. In addition, exposure to cold may produce certain **local effects**, *e.g.*, chilblains, or, in severe cases, frost-bite, the part affected becoming when frost-bitten, bloodless, ash-grey, and insensible. If a part affected with frost-bite is warmed too suddenly, gangrene is apt to set in, hence warmth should be restored to frost-bitten parts gradually, as for example, by friction with snow. **The principal circumstances modifying** the action on the system of exposure to cold are:—

1. **Movement of the atmosphere.**—Air being a bad conductor of heat, cold still air produces much less effect on the body, than cold air in motion.

2. **Moisture.**—If the surface of the body be wet, or covered with wet clothing, and exposed to cold air; heat, owing to evaporation, is withdrawn more rapidly than it the surface of the body be dry.

3. **Length of exposure.**—Of course the longer the exposure to

[1] Manual of Med. Jurisp. p. 462.

cold, the more likely are ill effects to occur. Adopting proper precautions, however, an extremely low atmospheric temperature may, as in the case of Arctic voyagers, be borne for long periods.

4. **Age.**—Adults bear cold better than the very young, and very old.

5. **Bodily condition of the individual.**—The action of cold on the system is favoured by anything which tends to lower the vital powers, *e.g.*, fatigue, exhaustion, intoxication, want of food, &c.

The principal post-mortem appearances met with in cases of death from cold are, according to Ogston:—(*a'*) **An arterial hue of the blood** generally, except when viewed in mass within the heart. (*b'*) **An unusual accumulation of blood on both sides of the heart,** and in the large thoracic arteries and veins. (*c'*) **Irregular diffused dusky-red patches** on limited portions of the surface of the body, even in the non-dependent parts (*frost erythems*). (*d'*) **Pallor of the surface of the body,** accompanied, according to Ogston, with anæmia, but, according to other authorities, with congestion of the viscera most largely supplied with blood. Ogston, however, found moderate congestion of the brain in three, and of the liver in seven, out of sixteen cases.

Death from cold is, as a rule, accidental.—Cases, however, are on record of children having been murdered by exposure to cold; and exposure to cold is a common method of infanticide in temperate climates (see *Infanticide*). Cases where insane persons have, it is alleged, been killed by exposure to cold, sometimes form the subject of a medico-legal inquiry. Taylor mentions a case where the death of a lunatic appears to have occurred from the combined effect of a shower-bath at 45° F. for half an hour, followed by a full dose of tartar emetic.

DEATH FROM DISCHARGE OF ELECTRICITY.

When a sudden discharge of electricity occurs through a bad or feeble conductor, accompanied by a spark or flash, disruptive discharge is said to have taken place. The discharge of atmospheric electricity by lightning; of sparks from an induction coil, or from the prime conductor of an electrical machine, are examples of this form of discharge. Disruptive discharge is attended by forcible mechanical separation of the particles of the bad or feeble conductor through which it occurs.

The human body is a feeble conductor of electricity; it allows of the passage through it, by conduction, of charges of electricity up to a certain pitch of intensity; but if this be exceeded, discharge taking place through the body becomes of the nature of a disruptive discharge. The passage of a feeble charge of electricity by conduction through the body usually

produces no ill-effects. A strong charge—strong enough to kill by shock—may pass through the body by conduction, or at any rate without producing visible separation of its particles. Hence, in some cases of death from discharge of electricity, no wounds can be discovered. Very strong charges of electricity discharge themselves through the body by disruption, producing visible wounds.

In addition to forcible mechanical separation of the particles of a bad or feeble conductor, three **other effects** may accompany electrical discharge, viz.: *a'*, Production of **chemical action**; (*b'*) Development of **heat**; and, (*c'*) Development of **magnetism.** In cases of death from lightning, the production of these effects, especially the two last, often affords strong confirmatory evidence as to the cause of death. Thus, in such cases, the skin or clothes often show marks of burning; metal articles attached to the clothes, such as buttons, or carried in the pockets, show signs of fusion; and steel articles, a knife for example, are found to have acquired magnetism.

Death or injury from discharge of atmospheric electricity (lightning stroke) has occurred in almost every situation. Thus persons have been struck by lightning in the open, in houses (in one case a boy in bed was struck by lightning), under trees, &c., &c. Not unfrequently, of two or three persons standing near one another, one is struck, the others escaping. During a thunderstorm, the neighbourhood of a high projecting bad or feeble conductor, such as a solitary tree, is a specially dangerous situation. The projecting object attracts the accumulated electricity, but, being a bad or feeble conductor, opposes such resistance to its passage, that lateral discharge takes place into neighbouring objects, *e.g.*, into the body of an individual standing near. The neighbourhood of a good conductor, if of insufficient thickness, is dangerous, for a precisely similar reason. Telegraph clerks, for example, have, during thunderstorms, been killed while standing at their instruments, owing to the wires in connection therewith, from their insufficient thickness, opposing so much resistance as to cause lateral discharge. The attraction of projecting objects for electricity necessitates the protection of high buildings by lightning conductors. These are thick rods of copper, one end of which projects above the building, while the other is buried in wet earth. All metal work on the surface of the building should be in electrical connection with the lightning conductor by thick wires. A peculiar class of cases of death resulting from the discharge of atmospheric electricity are the cases in which individuals are killed by what is called the "**return shock.**" In these cases the person killed is sometimes at a considerable distance from the spot where the discharge of lightning takes place. Cases of this kind are explained as follows. A cloud charged with electricity induces a charge of the opposite kind in objects

—*e.g.*, the bodies of individuals—in its neighbourhood. When the cloud discharges itself, the inducing influence being suddenly withdrawn, these objects suddenly discharge their induced charge of electricity. Sometimes this discharge of induced electricity from the body of an individual is so violent as to produce a severe or even fatal shock. In cases of this kind no marks of injury are found on the body of the sufferer.

Death or injury from electrical discharge may also occur from electricity other than atmospheric electricity. Powerful "arc" electric lamps (*i.e.*, lamps in which the light is produced by disruptive discharge between carbon terminals), require currents of great intensity. Two or three cases have lately been recorded where individuals have been killed by accidentally "short-circuiting" such currents through their bodies, *e.g.*, by grasping the wires conveying the current one in either hand, or by standing on one wire and laying hold of the other.

The effects produced on the body by the passage through it of an electrical discharge may be—1, Local; 2, Constitutional.

1. **The local effects** produced may be burns, blisters, or wounds; or ecchymosed streaks, spots, or patches. **Burns and blisters** are sometimes the result of the clothes having caught fire, but may occur independently of any ignition of the clothes. The hair is often found singed. If a **wound** is found it may be lacerated, punctured, or contused in character. **Ecchymosed** or **livid patches, spots,** or **streaks,** are frequently met with. Sometimes the streaks present a peculiar arborescent appearance. **Fractures** are rare, but have been found in a few cases (Tidy). No marks whatever may be found on the body, even in fatal cases.

2. **The constitutional effects** produced may be **immediate death** from shock; or the individual may fall down insensible, and **die after an interval** varying from a few minutes to several days. In one case death occurred as late as the thirty-third day after the receipt of the injury. If immediate death is not caused, the probabilities appear to be in favour of recovery taking place. In non-fatal cases **various nervous affections** have been found, *e.g.* paralysis (hemiplegia or paraplegia), loss of sight, hearing, speech, or memory.

The signs indicating death or injury from electric discharge may be:

1. **Marks on the surface of the body.**—The nature of these has been already described. The livid arborescent streaks found on the body in some cases are peculiarly characteristic of death from lightning stroke. The marks present on the body may simulate in appearance marks of mechanical violence.

2. **Internal appearances.**—Injury to the brain or its membranes is frequently found. The membranes may be congested or lacerated. The brain may be congested or disorganised.

Blood may be found effused on the surface or into the interior of the brain.

3. **Objects on or near the body** may show signs of the passage of electric discharge. The clothes may be found burnt or torn; the boots have sometimes been found burst open. In one case the whole of a man's clothes were torn off his body, and scattered about. Metal articles attached to the clothes or carried in the pockets may be found fused; and steel articles may be found to have become magnetic. Objects in the neighbourhood of the body may be found to show signs of injury; *e.g.*, a wall or building may be found cracked, or shattered and thrown down. Trees may be found split, and combustible objects, especially if dry, may be set on fire, or show marks of burning.

DEATH FROM STARVATION.

The essential nutritive constituents of food are: 1, **Albuminates**; 2, **Carbohydrates**; 3, **Fats**; and, 4, **Salts**. In order to maintain health and strength, a certain amount of each of these, plus a certain amount of water, must be daily supplied.

Of the essential nutritive constituents of food the albuminates, *e.g.*, albumen and casein, contain both carbon and nitrogen. The carbohydrates, *e.g.*, starch and sugar, contain carbon but no nitrogen. The fats, like the carbohydrates, contain no nitrogen, they, however, contain a larger percentage of carbon than the carbohydrates. For convenience, we may call the nitrogen contained in albuminates nutritive nitrogen, and the carbon contained in albuminates, carbohydrates, and fats, nutritive carbon.

The daily food requirements of the body depend (*a'*) **On the weight** (in health) of the individual to be fed. (*b'*) **On the amount of work** performed, and (*c'*) **On the age** of the individual, (children require more food in proportion to their weight than adults, seeing that in their case growth as well as nutrition must be provided for).

The daily food requirements of an adult may be stated as follows:—

1. **An adult requires daily if at rest** 25 grains, or if at work about 30 to 45 or 50 grains (according to the amount of work done) **of nutritive carbon** per 1 lb. of body weight.

2. **With this amount of carbon, nutritive nitrogen** must be supplied in amount equal to one-fifteenth to one-twentieth of the weight of the carbon.

3. **The food supplied must contain** a certain amount of **fat**, say about 1 to 3 ounces per diem.

4. **The food supplied must contain** a certain amount of the **salts**, *e.g.*, phosphates, required for the nutrition of the body. A certain amount of common salt, say ¼ to ½ an ounce, must also be daily supplied.

5. In addition a certain amount of **water**, 3 to 6 pints daily, and **condiments** are required.

6. Lastly, the food should be **varied** in character, of **good quality**, properly cooked, and the intervals between meals should not be too long.[1]

A rough rule for calculating the daily food requirements of adult natives of India is as follows:—Given (*a*) that the food consists solely of cereals and pulses fairly free from husk, and that the dietary contains a sufficiency of fat, and (*b*) that the amount of nutritive nitrogen in the dietary equals about one-twentieth of the carbon, then the number of ounces of food daily supplied must be not less than the average body weight in pounds, of the individuals to be fed, multiplied by

For bare subsistence, ·16.
For light work, say not over 1 foot-ton per 1 lb. of body weight, ·21.
For moderate work, say not over 2 foot-tons per 1 lb. of body weight, ·26.
For hard work, say up to 3 foot-tons per 1 lb. of body weight, ·31.

The rapidity with which ill effects follow deficient supply of nourishment, is affected by

1. **Age.**—The old bear deprivation of food better than adults, and adults bear deprivation of food better than children.

2. **Condition of body.**—Fat people bear deprivation of food best. Diminished activity of the vital functions (as in catalepsy), delays the occurrence of ill effects from deprivation of food (see *Case* A 43).

3. **Exposure to cold.**—Where the loss of heat from the surface of the body is rapid, the effects resulting from a deficient supply of the matters (food) required to maintain the normal temperature are more quickly felt than when the loss of heat from the surface is slow.

4. **Deprivation of water.**—Complete abstinence from both food and water kills more rapidly than abstinence from food alone. Taylor[2] states that it is probable, that in a healthy person under perfect abstinence (from both water and food), death would not commonly take place in a shorter period than a week or ten days. Guy[3] mentions a case of shipwreck, where of eighteen persons deprived of food and water, only one survived the eighteenth day. Where the abstinence is from food only, an individual may survive for a much longer period, thus a case is recorded of a madman who survived forty-seven days, and another of survival for sixty-one days: in the first of these two cases water only was taken, in the second water and a little orange-juice.

The Symptoms of Starvation.—The most prominent of these are:—

1. **Exhaustion, emaciation and loss of weight,** the sub-

[1] For tables showing the nutritive value of different articles of food, see *Appendix*, Tables X. to XII.
[2] Med. Jurisp. p. 141.
[3] For. Med. p. 345.

cutaneus fat disappears, and the muscles waste. The skin of the face becomes wrinkled, and that of the body, especially in previously plump persons, becomes baggy. Chossat, from a series of experiments on animals, found, as a rule, death to occur when the animal had lost two-fifths of its weight. Observation seems to indicate that this rule holds fairly good in the case of human beings.

2. **Thirst, pain and irritation of the stomach,** and **usually a costive condition** of the bowels. The outlets of the body are frequently found inflamed.

3. **The pulse** is at first quickened, but subsequently becomes slow. It usually, however, becomes greatly quickened on the approach of danger (Tidy).

4. **In chronic cases especially, the skin** frequently becomes covered with "a brown filthy-looking coating," and the body emits a fœtid odour. "**The gums** become swollen and ulcerated, and there is great tendency to ulceration and sloughing on the receipt of slight injuries" (Cornish).

5. **Delirium and convulsions** in some cases precede death, in other cases the mind remains unaffected.

The Post-mortem Appearances.—These are chiefly great emaciation, a shrunken and contracted condition of the stomach and intestines, a more or less atrophied condition of the viscera, and absence—not necessarily complete in acute cases—(see *Case* A 47) of fat. It should be noted, however, that all these appearances may be present in death from exhausting diseases. Hence in cases of death from alleged homicidal starvation, the body should be carefully examined for appearances indicating the existence of such diseases. It may, in such cases, be an extremely difficult matter to form a definite opinion as to whether death was due to disease or starvation. (For a case in which this question arose, see *Case* A 44).

Starvation may be accidental, homicidal, or suicidal.—The most common causes of **accidental starvation** are: 1. Shipwreck, as in the case just quoted; 2. Mining accidents—individuals by a fall of earth getting shut up in a mine; 3. Disease, *e.g.* stricture of the œsophagus; and 4. Famine. **In homicidal cases** the victim is usually an infant or child. The withholding of food with or without exposure to cold is a not infrequent method of infanticide (see *Infanticide*). Cases also are not infrequent where children have been starved by their parents or other persons having charge of them. In fatal cases of this kind, as already pointed out, the body should be carefully examined for signs of disease. In non-fatal cases, an unusually low body weight, coupled with a rapid gain in weight when proper nourishment is administered, is very strong evidence in favour of starvation (see *Case* A 45). As already pointed out under suffocation, in one form of "Samádh," or burial alive of

lepers, the head is left uncovered, and death takes place from exhaustion, the result of starvation and exposure; and not—as in cases where the burial is complete—from suffocation. **Suicidal cases** are rare, but are sometimes met with, especially in the insane. Prisoners sometimes also attempt to commit suicide by starving themselves.

Pretended fasting.—Cases are on record where individuals, as a rule hysterical girls, or young women, have pretended to an ability to abstain for long periods from food (see *Cases* A 46 and 47). A medical man should in such cases be cautious about undertaking the duty of watching the impostor with a view to detection, as if death results he may be held criminally responsible. In the case of the Welsh fasting-girl (*Case* A 47), the medical men who had accepted the responsibility of superintending the watching were indicted before the magistrates along with the parents of the girl, the parents only, however, were committed for trial.

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CHAPTER VII.

GENERAL TOXICOLOGY.

UNDER this head the following general questions relating to poisons may be considered:—I. The definition of the term "a poison," and the law relating to poisons. II. The statistics of poisoning. III. The mode of action of poisons. IV. The classification of poisons. V. The treatment in cases of poisoning; and, VI. The evidence of poisoning.

I. THE DEFINITION OF THE TERM A POISON, AND THE LAW RELATING TO POISONS.

The exact definition of the term "a poison" is a matter of considerable difficulty, chiefly because:

1. **All substances** which are **capable of causing death** when absorbed into the system **are not poisons** in the ordinary acceptation of the term. Thus, some substances, *e.g.*, **common salt, and sulphate of potash,** are harmless in small doses, and cannot properly be called poisons, yet either of these, if administered in sufficiently large quantity, may cause death. Again, **morbid poisons** are matters which, when absorbed into the system, are capable of causing death, but are not poisons in the ordinary acceptation of the term.

2. **Again, some substances** which, when swallowed, are capable of causing injury or death, **produce their effect independently of absorption into the system,** *e.g.*, **the corrosive acids and mechanical irritants.** Any complete definition of the term "a poison" ought, however, to include such substances, notwithstanding the fact that they are capable of producing ill effects, independently of absorption into the system.

For medico-legal purposes in India, the **exact definition** of the term "a poison" is **not a matter of much importance,** for the following reasons:—

1. **Sect. 299 of the Indian Penal Code** declares that: "Whoever causes death by doing an act with the intention of causing death, or with the intention of causing such bodily injury as is likely to cause death, or with the knowledge that he is likely by such act to cause death, commits the offence of **culpable homicide.**" Hence, if A. administers a substance to B., with such

intent or knowledge, and thereby causes B.'s death, A. may be convicted under this section, of the offence of committing culpable homicide, irrespective of whether the substance administered may or may not strictly be called a poison

2. **Again, sections 324 and 326** (already referred to, see *Wounds*, page 40, and *Burns*, page 92), make the causing of **hurt**, and **grievous hurt**, by certain specified means, offences of greater gravity than the causing of respectively hurt or grievous hurt by means other than those specified in these two sections. Among the means specified in the two sections in question, we find: "**any poison or any corrosive substance,**" or "**any substance which it is deleterious to the human body to inhale, to swallow, or to take into the blood.**" Hence, for conviction under these sections, it is not necessary to establish that the substance by means of which the hurt or grievous hurt was caused is a poison; it is sufficient if it be proved that it is a substance which comes under the above-stated description.

It is possible, however, that the question of the exact **definition** of the term a "poisonous substance" might arise in the case of a person charged **under section 284 of the Penal Code** with the offence of "knowingly or negligently" omitting "to take such order with any poisonous substance in his possession as is sufficient to guard against probable danger to human life from such poisonous substance."

One other section of the Penal Code relating to the administration of poisons may be here referred to. **Section 328** states that: "Whoever administers to, or causes to be taken by, any person, any poison or any stupefying, intoxicating, or unwholesome drug or other thing, with intent to cause hurt to such person, or with intent to commit, or to facilitate the commission of an offence, or knowing it to be likely that he will thereby cause hurt, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine." Here again it will be observed that the addition of the words "any stupefying, intoxicating, or unwholesome drug or other thing," render the exact definition of the term "a poison" unnecessary for the purposes of this section. It should, however, be noted that the words "or other thing" must be read "other unwholesome thing." "Hence, administering a substance as to whose nature no evidence was given, which was intended to act as a charm, was held to be no offence (*R.* v. *Jotee Ghoraee*, 1 Suth. Cr. 7)."[1]

Legal restrictions are placed on the sale of poisons—(a) In England, by, The Arsenic Act (14 Vict. cap. 13); and The Pharmacy Act (31 & 32 Vict. cap. 21); the chief provisions of which are:—(1) That except in special cases, arsenic (*i.e.* arsenious oxide) is only to be sold mixed with soot or indigo, in

[1] Mayne's Penal Code, p. 282.

the proportion of one ounce of soot or half an ounce of indigo at the least, to one pound of the arsenic; and, (2) The restriction of the privilege of retailing poisons to certain persons, who must enter each sale with the purchaser's name and address, in a book kept for the purpose.

(b) **In the Presidency of Bombay,** by The Sale of Poisons Act (Bombay Act VIII. of 1866). The chief provisions of this Act are that certain poisons named in Schedule A of the Act—(1) May only be sold by licensed vendors (sect. 3). (2) (Except when dispensed as medicine on the order or prescription of a practitioner of medicine) may only be retailed to persons known to the vendor, or in presence of a witness known to the vendor, and to whom the purchaser is also known; and each sale must be entered with the purchaser's name and address in a book kept for the purpose (sects. 13, 14 and 19); and (3) Pounded white arsenic (except in special cases) may only be sold mixed with soot in the proportion of one ounce to each pound of arsenic, or with indigo or Prussian blue in the proportion of half an ounce to each pound of arsenic (sect. 17).

Schedule A of the Act enumerating the poisons to which it applies contains the following list of poisons:—"1. **Vegetable poisons.**—Aconite (*butchnag*), cocculus indicus (*kakmari, kakphul*), datura (*dhatoora*), henbane (*khorasanee ajwan*), nux vomica (*koochila and kajra*), Saint Ignatius' bean (*papeeta*), and Calabar bean. 2. **Mineral poisons.**—White arsenic (*phutkya somul, somul*), red arsenic or realgar (*munseel*), yellow arsenic or orpiment (*hurtal*), Scheele's green or arsenite of copper, and Schweinfurth green or aceto-arsenite of copper (*hirwa*), and corrosive sublimate (*ruskapoor*)."

II. THE STATISTICS OF POISONING.

From the tables contained in the Appendix[1] it will be seen that of the total deaths from violence occurring yearly in England, about 1¼ per cent., or 8·77 per million of population are due to poison. In the Bombay Presidency the deaths from poison recorded yearly correspond to about 6·80 per million of population, or about 1¾ per cent. of the total deaths from violence in each year. The medico-legal returns of Bengal, &c.,[2] for the three years ending 1872 showed a total of 1140 deaths from poison, equal to 380 per annum.

In England, during the five years ending 1867, more than two-thirds of the total deaths from poison were due to opium, lead-salts and cyanides; these poisons accounting respectively for 41·4, 15·9 and 11·3 per cent. of the cases. Of the remainder,

[1] Tables XIV. and XV., Appendix, p. 501.

[2] Including the N.W. Provinces, Oudh, Central Provinces, and Punjaub (Harvey's Report, p. 245).

about two-thirds were due to the following poisons, mentioned in order of frequency of employment: arsenic, oxalic acid, mineral acids, mercury, and strychnia.

In India, the two poisons most frequently used are **arsenic** and **opium.** During the year 1884, for example, over 90 per cent. of the deaths from poison reported to the chemical examiners, were due to one or other of these poisons. Again, during the ten years ending 1884, these two poisons between them accounted for over 72 per cent. of the cases, and more than 87 per cent. of the deaths from poison, reported to the Bombay Chemical Examiner. Of the two poisons just mentioned, arsenic, usually in the form of arsenious oxide, is used both for homicidal and suicidal purposes, and largely also as a cattle poison. As regards opium-poisoning, this in adults is almost always suicidal, but in young children accidental cases are frequently met with. Opium, in fact, is seldom used homicidally except in cases of infanticide. Next to arsenic and opium, the poison probably most frequently employed in India is **dhatura**; this, however, is mainly employed for purposes of drugging, with the object of facilitating the commission of an offence, usually theft. Other poisons less frequently used in India, but still of sufficiently frequent use to deserve special mention, are as follows:—

1. **Mineral poisons.**—(*a*) **Copper.** Cases of poisoning by compounds of this metal frequently arise accidentally from contamination of food by the copper cooking-vessels largely employed in India; occasionally, however, cases of attempt at homicide by the administration of sulphate of copper, are met with. (*b*) **Lead.** Chronic poisoning by lead, common in England, is rare in India. Red lead, however, is tolerably frequently met with in India as an ingredient of local applications employed to procure abortion. (*c*) **Mercury.** Cases of chronic mercurial poisoning arising from malpraxis, although not met with so often now as formerly, are still occasionally seen. Occasionally also ruskapoor, or calomel containing a variable percentage of corrosive sublimate, is criminally employed. (*d*) **Pounded glass.** This is popularly believed in India to be a powerful poison, and is occasionally mixed with food with homicidal intent.

2. **Vegetable poisons.**—Next to **opium** and **dhatura,** the vegetable poisons most frequently used for homicidal and suicidal purposes are **aconite,** nerium odorum or **oleander** (and cerbera thevetia), **nux vomica** (and its alkaloid strychnia) and various **euphorbias.** Of these, aconite and strychnia have also given rise occasionally to accidental cases, notably in the case of the first, from its use as a fortifying agent for alcoholic liquor, and in the case of the second from its use as a dog-poison. The seeds of cerbera thevetia, it may be mentioned, are sometimes used as a cattle-poison, and the milky juice of the milk bush, and other

euphorbias, as an ingredient in irritant preparations employed as local applications for the purpose of procuring abortion. Another vegetable poison often used for this last purpose is **plumbago rosea**, and occasionally various **cucurbitaceous tubers** are internally administered with the same object. Lastly, it may be mentioned that **mudar** (calotropis procera) and **tobacco** are said to be used for purposes of infanticide, and that in some parts of India a paste made from the seeds of **abrus precatorius** is used, by subcutaneous insertion, for the purpose of destroying cattle.

III. THE MODE OF ACTION OF POISONS.

A poison may produce its effects owing to its having been administered by the mouth, inhaled into the lungs, absorbed through the skin, injected into a wound, or introduced into the rectum, or vagina. A case even is recorded of death from the pouring of a corrosive poison (nitric acid) into the ear during sleep. **The action of a poison** may be (*a'*) local, or (*b'*) remote, and the same poison may possess both a local and a remote action. **The local action** of a poison may consist in the production of (1) **Corrosion**, *i.e.*, chemical destruction, as in the case of the strong mineral acids. (2) **Irritation and inflammation**, as in the case of cantharides, tartar emetic, &c.; or (3) **Certain nervous impressions**, as in the case of opium, aconite, &c.

The remote action of a poison may be either of a **non-specific** or **specific** character.

Non-specific remote action.—Poisons which possess a violent local action, sometimes exert a remote non-specific action on the system, producing thereby an effect similar to that which often results from severe mechanical injury. Extensive corrosion produced by a corrosive acid, may, for example, be followed by shock, as a remote non-specific action.

Specific remote action.—This varies in character; it may, for example, consist in the production of tetanic spasm, as in poisoning by strychnia; syncope, as in poisoning by tobacco; nephritis, as in poisoning by cantharides; gastritis, as in poisoning by arsenic, &c., &c.

The local action of a poison **results from its direct application** to the part affected. **The remote specific action** of a poison **results from the absorption** of the poison into the system through the blood. Absorption takes place with extreme rapidity. Blake, from his experiments, inferred that a poison might be diffused through the whole body in nine seconds; and Erichsen, in a case of extroversion of the bladder, found potassium ferrocyanide in the urine, one minute after it had been given by the mouth on an empty stomach.

The fact that poisons are absorbed into the system **through the blood**, has certain **practical bearings**. For example, it

indicates the advisability, in the case of poisoned wounds, of applying a ligature above the wounded part, and endeavouring to remove the poison from the wound by excision and suction. Again, it indicates that after death, absorbed poisons will probably be found in greatest quantity, in organs containing much blood, *e.g.*, the liver.

There is some reason to suppose that remote action may in some cases be the result of "**sympathy**," that is, of an impression conveyed to the nerve-centres by the nerves. This view receives support from the fact that in experimenting on animals with hydrocyanic acid, death has been found to occur with extreme rapidity. Dogs, for example, have been killed in two seconds by a dose of this poison.

Certain causes may modify the action of a poison. The chief of these are :—

1. **Quantity administered.**—The administration of a large dose of some poisons is sometimes followed by symptoms differing greatly in character from those which follow a moderate dose; *e.g.*, moderate doses of arsenic produce irritant symptoms, very large doses sometimes cause death by shock without irritant symptoms.

2. **Difference in form.**—(1) **Physical difference.** Poisons act most rapidly when gaseous; next, when liquid; next, if in fine powder; and least rapidly when in solid masses (see *Case* B 13). (2) **Chemical difference.** This may have the effect—(*a*) **If the poison acts chemically** only, of rendering an active poison inert, *e.g.*, corrosive acids may be rendered inert by combination with alkalies; or (*b*) **If the poison does not act chemically** only, chemical difference in form may, by rendering the poison more soluble, increase the rapidity with which it acts, or, by rendering it less soluble, diminish the rapidity of its action.

3. **Mechanical mixture** with inert substances, *e.g.*, dilution, or mixture with inert powders.—In some cases this **alters the character of the symptoms** produced; for example, corrosive acids, when diluted, act as irritants only. In other cases, mechanical mixture with an inert substance, by protecting the poison from absorption, may **delay its action**; hence poisons, as a rule, act less rapidly when given on a **full stomach**. Again, **animal charcoal**, by taking up a poisonous alkaloid and rendering it **insoluble by adhesion**, may delay or prevent its action.

4. **Mode of introduction.**—This, by affecting rapidity of absorption, affects the rapidity of action of poisons. Modes of introduction enumerated in order of rapidity of action, the most rapid first, are (1) injection into a vein; (2) application to a wound; (3) application to a serous surface; (4) application to the broncho-tracheal mucous membrane; (5) introduction into the stomach; (6) injection into the rectum, and (7) application to the unbroken skin.

5. **Habit.**—This, in the case of many poisons, *e.g.*, opium, alcohol, and tobacco, tends to confer on the system a resisting power to the action of the poison to the use of which the individual is habituated.

6. **Idiosyncrasy.**—This may show itself either in abnormal sensitiveness to the action of a particular poison,—*e.g.*, mercury, —or the reverse.

7. **Existence of disease.**—This, if the symptoms of the disease resemble those produced by the poison, tends to confer increased sensitiveness to the action of the poison: *e.g.*, narcotics in advanced renal disease. If, on the other hand, the symptoms of the disease are opposed in character to those produced by the poison, diminished sensitiveness to the action of the poison may be the result: *e.g.*, narcotics in tetanus.

8. **Sleep and intoxication.**—These may delay the action of a poison (see *Cases* B 10 *a'*, *b'*, and *d'*).

9. **Accumulation.**—Small doses of a poison, each insufficient to cause any serious effect, if given one after the other at short intervals, may accumulate in the system and produce serious effects. Accumulation obviously tends to occur, when the rate of elimination of the poison is slower than the rate of its administration. Hence poisons which are only slowly eliminated from the body,—*e.g.*, lead and mercury, and metallic poisons generally,—are specially prone to act as cumulative poisons. Organic poisons are, as a rule, quickly eliminated. In some, however,—*e.g.*, strychnia—the rate of elimination is comparatively slow, and accumulation tends to occur.

IV. THE CLASSIFICATION OF POISONS.

Poisons may be classed according to their origin: *e.g.*, mineral, vegetable, &c., or according to their action. Adopting the latter method, we find that some poisons possess a marked local irritant action, and are therefore called irritant poisons. Others possess little or no local irritant action, but exert their influence on the nervous system, and may therefore be called neurotic poisons. Of the neurotic poisons, some—cerebral poisons—act mainly on the brain; others—spinal poisons—act mainly on the spinal system; and others—cardiac poisons—act mainly on the heart. Again, some poisons appear to act by destroying the vitality of the blood; these are called septic poisons. Lastly, we have poisons which give rise to disease or morbid poisons. Excluding morbid poisons, the study of which does not belong to the subject of Forensic Medicine, it is possible therefore to divide poisons according to their action into five orders, viz :—

I. **Irritant poisons,** or such as possess a marked local irritant action, exciting irritation and inflammation; and when swallowed cause vomiting, and, as a rule, also purging. A few of these,

when concentrated, act as corrosives: *i.e.*, cause chemical destruction of the tissues.

II. **Cerebral poisons**, or such as act mainly on the brain, causing delirium or narcotism; and tend, as a rule, to cause death by coma.

III. **Spinal poisons**, or such as act chiefly on the spinal system, causing either tetanic spasm, or local anæsthesia or hyperæsthesia, or paralysis; and tend, as a rule, to cause death by asphyxia, from spasm or paralysis of the respiratory muscles.

IV. **Cardiac poisons**, or such as act mainly on the heart, and tend to cause death by syncope.

V. **Septic poisons**, or such as are supposed to act by destroying the vitality of the blood; a few only of these, however, properly belong to the subject of Forensic Medicine.

Certain of the above groups, or orders, notably the two first-mentioned, include a large number of poisons, and may be, therefore, conveniently divided into classes and sub-classes. Many poisons, it must also be noted, possess more than one of the above described actions, and may therefore be classed in more than one of the above five orders. Thus, for example, some poisons possess both a marked local irritant action, and a specific remote action on the nervous system. Such poisons may be called compound irritants, or neuro-irritants, and those of them that act on the brain may be called narcotico-irritants. Some neurotic poisons, again, have a marked action on both the brain and spinal system, or on both the brain and the heart, and hence arise the terms cerebro-spinal and cerebro-cardiac poisons.

V. THE TREATMENT IN CASES OF POISONING.

The general indications of treatment in cases of poisoning are:—(1) Elimination; (2) Prevention of action; and (3) Counter-action and removal of effects.

1. **Elimination.**—The measures to be adopted for the purpose of procuring elimination of a poison, vary with the mode in which the poison has been administered. Thus, if the poison has been injected into a wound, **excision** of the wounded part and **suction** are indicated. If the poison has been inhaled into the lungs, the patient must be made to **inhale pure air**, so that the poison may thereby be chased out of the lungs. If the poison has been taken into the stomach, **vomiting should be promoted** by the administration of warm water, or set up by tickling the fauces, or by the administration of **emetics**; *e.g.*, mustard and water, or 20 to 30 grain doses of sulphate of zinc, or of powdered ipecacuanha;[1] or except in cases of corrosive poisoning the **stomach-pump** may be used, care being taken to inject warm water first before proceeding to exhaust, and to always remove a

[1] See also Apomorphine, p. 189.

little less than the quantity injected. **Special treatment** may be necessary for the elimination of absorbed poisons, *e.g.*, the administration of potassium iodide, in cases of poisoning by lead.

2. **Prevention of action.**—The nature of the measures by which this indication is carried out, vary with the nature of the poison, thus:—

(1) **Except in cases where the action of the poison is chemical or mechanical** in character, the measures indicated for adoption are measures calculated to prevent absorption of the poison. **Prevention of absorption** may be effected—(*a'*) **By the administration of substances which will render the poison insoluble,** *e.g.*, albumen in cases of poisoning by corrosive sublimate; freshly prepared hydrated ferric oxide in cases of poisoning by arsenic; sulphates in cases of poisoning by lead, &c., &c.: or (*b'*) **By the adoption of measures** calculated **to destroy** the poison, *e.g.*, cauterization of poisoned wounds; inhalation of chlorine, in cases of poisoning by sulphuretted hydrogen: or (*c'*) **By mechanical means,** *e.g.*, the application of a ligature above the wounded part, in cases of poisoned wounds.

(2) **Where the main danger** to be apprehended from the action of a poison, is **corrosion or chemical destruction** of the tissues with which it comes in contact, this second indication of treatment is carried out, by the administration of substances calculated to prevent the action of the poison, by entering into chemical combination with it; *e.g.*, the administration of alkalies in cases of poisoning by the corrosive acids.

(3) **Where the substance is one which acts mechanically** only, it is in some cases possible to prevent its action, by the administration of matters which will mechanically protect the tissues, from the action of the substance swallowed: *e.g.*, the administration of bulky food, in cases where pounded glass has been taken.

Substances which prevent the action of poisons by chemically destroying, or neutralizing them, or rendering them insoluble, are called **chemical antidotes.**

3. **Counteraction and removal of effects.**—In some cases of poisoning this indication may be carried out, by the administration of **physiological antidotes** or substances which exert an action on the system, opposed to that of the poison; *e.g.*, atropia in poisoning by opium. In other cases this indication may be carried out, by the adoption of various measures of treatment calculated to counteract or remove the effects of the poison; *e.g.*, the use of cold affusion and galvanism in narcotic poisoning; of warmth to the surface, stimulants, and the recumbent posture, in cardiac poisoning; of artificial respiration in cases where the poison taken is one which, like conium, tends to cause death by paralysing the respiratory movements; of demulcents in irritant

poisoning, &c. &c. Special measures adopted for the purpose of promoting the elimination of absorbed poison, already referred to under Elimination, may also be included under the head of measures directed to the removal of the effects of the poison.

VI. THE EVIDENCE OF POISONING.

Irrespective of non-expert evidence, evidence pointing to the administration of poison may be derived from the characters of: 1. The symptoms; 2. The post-mortem appearances; and from the results of, 3, Chemical analysis; and, 4, Experiments on animals.

1. The Symptoms.

These possess certain general characters, viz.:

1. **They suddenly arise.**—This character however may be absent in a case of poisoning, *e.g.*, in chronic poisoning by lead, mercury, phosphorus, &c.; and may be present in cases not of poisoning, *e.g.*, apoplexy, cholera, &c.

2. **They steadily increase in severity.**—This character, like the last, is often present in disease. Again in some cases of poisoning, this character is absent, *e.g.*, in the remittent form of opium poisoning (see *Case* B 1); and in cases where small doses of a poison are administered at short intervals.

3. **They are uniform in character,** *i.e.*, with the known effects of a particular poison; hence gastritis followed by salivation, as in acute mercurial poisoning, or by paralysis as in arsenical poisoning, do not form exceptions to this rule.

4. **They commence soon after taking food, drink, or medicine.**—This character may be absent owing to the symptoms of poisoning being delayed in their appearance by sleep, or by intoxication (see *Cases* B 10, *a'*, *b'* and *d'*); or by the counteractive effects of another poison simultaneously administered. Or again this character may be absent, owing to the nature of the poison swallowed; for example, sparingly soluble lead salts only give rise to acute symptoms after an interval of several hours, and a similar interval is often noticed in cases of fish poisoning. This character also may be present in cases not of poisoning. Cholera, apoplexy, &c., may come on soon after a meal, or rupture of the stomach may occur (see *Case* B 6); and symptoms closely resembling those of poisoning, have appeared as the result of swallowing, after exertion, a quantity of cold fluid (see *post*).

5. **Others are affected who partook of the same food, &c.** This is a very striking character; it may however be present in disease, *e.g.*, where, as sometimes happens, several persons after partaking of a meal together, are nearly simultaneously attacked by cholera. This character may be apparently absent in a case of poisoning, *e.g.*, where (as in *Case* B 2), of several persons present

at a meal, only one partakes of a particular dish. Poisoning also may be indicated by the fact that several persons have suffered from suspicious symptoms, after partaking of articles of food, &c., which have passed through the hands of one and the same individual; although the attacks occurred at different places, and at different times (see *Case* B 3).

6. **They appear in persons previously in good health.**—This character may obviously be absent in cases of poisoning, or present in cases of disease; and

7. **They prove rapidly fatal.**—This character, like the last, is one which may be absent in poisoning, and present in disease.

Obviously the greater the number of the above characters present in the same case, the stronger is the suspicion of poisoning; and *vice versâ*, the smaller the number, the weaker the indication of poisoning.

The special characters of the symptoms of poisoning vary with the class to which the poison belongs. Certain affections are accompanied by symptoms more or less resembling in special character those of poisoning.

1. **Irritant poisons.**—The chief affections simulating the effects of poisons of this class, are:

(*a*) **Cholera.**—This is specially liable to be mistaken for arsenical poisoning, and *vice versâ* (see *Arsenic*, p. 144).

(*b*) **Gastritis,** following the imbibition of a large quantity of cold fluid, whilst the body is cooling after violent exertion (see *Case* B 5). Or, under such circumstances, death may occur from shock (see *Case* B 4), and the case resemble one of rapidly fatal narcotic poisoning, *e.g.*, by hydrocyanic acid. Idiopathic gastritis is very rare, and is not accompanied by the violent purging usually present in irritant poisoning.

(*c*) **Rupture of the stomach,** complete or partial, especially when due to over-distension (see *Case* B 6), may closely simulate irritant poisoning. So also may perforation of the stomach from disease, rupture or perforation of the intestines, and rupture of the biliary ducts, uterus, or uterine appendages. In cases such as these the post-mortem appearances will indicate to what the symptoms have been due.

(*d*) **Colic.**—There may be some difficulty in diagnosing this from acute irritant poisoning, especially by lead salts. Pressure, however, in acute irritant poisoning augments the pain, while in colic it often relieves it.

(*e*) **Enteritis, Peritonitis, and Intussusception.**—These affections, like acute poisoning by lead salts, are accompanied by constipation. Unlike irritant poisoning, in the later stages of these affections, vomiting, if present, becomes stercoraceous.

2. **Cerebral poisons.**—The chief affections simulating the effects of these, are:—

(*a*) **Apoplexy and uræmic coma.**—These may more or less

resemble poisoning by opium, or narcotics similar in action thereto (see *Opium*, p. 255).

(*b*) **Epilepsy.**—A fatal attack of this affection might possibly be mistaken for hydrocyanic acid poisoning. Death, however, seldom results from a first attack of epilepsy, and a history of previous attacks would most probably indicate the true nature of the case.

(*c*) **Sudden death from heart disease.**—This may be mistaken for hydrocyanic acid poisoning, or for one of those cases which sometimes occur, of death by syncope from a single overdose of chloral. The presence of post-mortem appearances of advanced heart disease, would of course tend to indicate death from disease. In some cases of sudden death from heart affection, however, no marked appearances of disease of the heart are discoverable after death.

3. **Spinal poisons.**—The effects of poisons of this class may be more or less simulated by

(*a*) **Tetanus.**—This closely resembles strychnia poisoning (see p. 277).

(*b*) **Cerebro-spinal meningitis.**—This affection is accompanied by tetanic spasms, more or less resembling those of strychnia poisoning. Unlike strychnia poisoning, headache, fever, hyperæsthesia, and delirium precede the tetanic symptoms.

(*c*) **Convulsions** in young children sometimes prove rapidly fatal. Such cases may simulate poisoning, the more so as opium poisoning in children is often accompanied by convulsions. In some cases the attack may be traced to dentition, indigestion, worms, or other source of irritation, but sometimes no cause for the attack is discoverable.

4. **Cardiac poisons.**—The effects of a poison of this class may be simulated by heart disease (see above), or by sudden death from embolism, especially of the pulmonary artery. In this last case, the discovery of a plug obstructing the affected vessel, indicates the cause of death.

2. The Post-Mortem Appearances.

A large number of poisons leave no characteristic post-mortem appearances. Irritant poisons, however, usually leave well-marked signs of their action. Such signs may consist in the presence of—

1. **Redness of the mucous membrane of the stomach** and other portions of the alimentary canal, due to inflammatory action. Such redness may be the result of disease, but is usually the result of the administration of an irritant poison. When due to poisoning, the redness may vary in degree from unusual vascularity, to a deep red velvety appearance. The mucous membrane is softened and opaque, and may show dark patches, due to underlying extravasated blood. Often its surface is covered

with a glairy tenacious mucus, in which particles of the poison may be found entangled. Usually, in irritant poisoning, these appearances are chiefly met with in the stomach. They may, however, extend to the duodenum, or beyond. Redness due to inflammatory action may be more or less simulated by—(*a*) **Staining with red dyes.**—Chemical tests will usually distinguish this, most vegetable reds being turned either blue or green by alkalies, or yellow by acids. Examination under the microscope, also, will in such cases show that the redness is not due to distension of the blood vessels. (*b*) **Congestion not due to irritation.**—In some cases of sudden death, especially from congestion of the brain, or from apnœa, the mucous membrane of the stomach is found congested, and patches even of extravasated blood have been found beneath it. In such cases, as a rule, the redness is only a diffused blush, not stellated or punctated, as in irritant poisoning. On dissection, also, the mucous membrane is found to be tough and transparent, not softened and opaque from inflammation, as in irritant poisoning.

2. **Discolorations other than redness** of the parts with which the poison has come into contact. In some cases such discolorations are met with in the alimentary canal. Thus, in arsenical poisoning, yellow patches, due to conversion of arsenious oxide into sulphide, are often found on the mucous membrane; and in cases of copper poisoning a blue or green coloration may be found. In cases of corrosive poisoning also, discolorations varying according to the poison may be found, not only on the mucous membrane of the alimentary canal, but also on other parts, *e.g.*, the skin.

3. **Ulceration of the mucous membrane of the stomach.**—Ulceration from disease must not be mistaken for this. Generally, but not always, in disease, the ulcer is only just surrounded by redness, the symptoms are slight, and, unless due to malignant disease, the individuals affected are generally young women, from eighteen to twenty-three years of age. In irritant poisoning the redness, as a rule, is diffused over the whole stomach, particles of the poison may be found adhering to the ulcer, the ulceration may extend into the duodenum, and the symptoms are severe.

4. **Corrosion or chemical destruction of the tissues, and perforation of the stomach.**—In cases of corrosive poisoning marks of corrosion may be found on the skin, or in the mouth, throat, or œsophagus, or on the mucous membrane of the stomach. Perforation of the stomach may be found; this, however, is comparatively rare in poisoning. Post-mortem softening of the stomach, with or without perforation, due to the action of the gastric juice, is sometimes met with, and must not be mistaken for corrosion. In such post-mortem softening, dependent parts of the stomach, and sometimes neighbouring organs, are affected.

There is no inflammatory redness, and the mucous membrane is gelatinous and transparent. The extent of the softening, also, is likely to be greater the longer the period which has elapsed since death.

5. Post-mortem appearances of **irritation** may also be found in the **air passages**, in cases of poisoning by volatile or gaseous irritants, *e.g.*, ammonia and hydrochloric acid; and in the case of certain irritant poisons, *e.g.*, cantharides, in the kidneys or **urinary passages**. **A yellow tinge of the skin** is a common post-mortem appearance in acute poisoning by copper and phosphorus, and in the latter, fatty degeneration of the liver is almost always present.

In the case of some non-irritant poisons, *e.g.*, hydrocyanic acid, the presence, on opening the body, of a particular **odour** may indicate the nature of the case. In others, during the post-mortem examination, **portions of the poison** used, *e.g.*, dhatura seeds, may possibly be found and identified. In the great majority, however, the post-mortem appearances present, if any, are merely such as indicate the "mode" of death,[1] and are therefore consistent with death from causes other than poisoning.

In making a post-mortem examination in a case of suspected poisoning, certain points require special attention.[2] The chief of these are:—

1. **The surface and orifices of the body, especially the mouth and throat, should be examined for marks of corrosion.**—This is most important. It frequently happens that in corrosive poisoning, chemical analysis can do no more than prove the existence in the viscera of a salt, *e.g.*, a sulphate or an oxalate, which may have been derived from the poison swallowed, or may have been introduced into the body as a constituent of an article of food or medicine. In such a case failure to examine the mouth and throat, for marks of corrosion, may make it impossible to prove that death was due to poison.

2. **The mucous membrane of the stomach and alimentary canal should be examined** at the time the post-mortem inspection is made. Appearances indicative of the action of a poison are liable (from decomposition, or from the action of preservative fluids) to disappear from the mucous membrane. Hence, after removal of the stomach and intestines, these should be cut open, and their internal appearance noted. Suspicious particles found adhering to the mucous membrane of the stomach should be picked off, and preserved separately.

3. **The preservation of matters for analysis.**—In addition to the stomach, its contents, and the contents of the intestines,

[1] Coma, Apnœa, &c.

[2] See Rules published by the Government of Bombay for the guidance of medical officers forwarding articles for analysis in cases of suspected poisoning, *Appendix*, XIII. p. 499.

one kidney, and a portion of the liver at least 1 lb. in weight, should always be preserved. Failure to preserve a portion, or a sufficient portion, of the solid viscera, may result in entire failure of the chemical analysis (see *post, Chemical Analysis*). Obviously thoroughly clean vessels alone should be used. For the purpose of preventing decomposition, spirit should be added to the matters preserved (except, of course, to fluid matters, in cases of suspected alcoholic poisoning). It is desirable to retain a sample of the spirit used, in case any question should arise in regard to its purity. The vessels containing the matters preserved for analysis should be sealed, and care taken to prevent their being tampered with.

4. **Transmission of articles for analysis.** — To secure identity, the containing vessels should be properly labelled, and an impression of the seal used in closing them (which, of course, should be a private seal), enclosed in the letter advising their despatch. The box containing the vessels should be franked. A summary of the case should always be forwarded to the analyst. It must be recollected that the quantity of matter available for analysis is limited, and that the quantity of poison present is frequently very small. Subdivision, therefore, of the matters under examination is to be avoided as much as possible, and this cannot be the case if the analyst has no guide to the class of poison to be searched for.

5. **General examination of the body.**—This should never be neglected. It should be remembered that even in cases where the suspicion of poisoning is strong, death may have been due to causes other than the administration of poison. Also that post-mortem appearances, indicative of disease or injury, may be found co-existing with appearances indicating death from poison; and that in such cases the fact of the existence of the disease or injury may, even when death has been clearly due to poison, be important as bearing on the question of suicide or homicide. In the case of female bodies, care should always be taken to examine the vagina. Poisonous matters, or traces left by their action, are frequently found in the vagina, in cases where death has been the result of an attempt to procure abortion. Even also in other cases poison may be found in the vagina (see *opium poisoning*, p. 254). The precautions to be attended to, &c., when examining the bodies of cattle, in cases of suspected cattle-poisoning, will be found in the Appendix (XVI., p. 504).

3. The Chemical Analysis.

It may first be pointed out that poisons, after absorption, tend to undergo elimination by natural effort, *e.g.*, by the lungs, skin, or kidneys. Hence, during life, in cases of poisoning, poisons may be detected by analysis in the urine. Hence, also, if in a

case of poisoning, life is prolonged for some time, no poison may, after death, be discoverable in the body. The longer life is prolonged, and the more soluble or volatile the poison, the more likely is this to occur. Complete elimination has been known to take place, in a case of arsenical poisoning, in a fortnight (see *Case* B, 25*a*); and, in a case of antimonial poisoning, in a week; and may occur very rapidly in the case of very volatile poisons, like hydrocyanic acid and chloroform.

Poison may be detected by analysis:—(*a'*) **Before death,** in the vomit, urine, or in other evacuations; or in food, or other suspected articles. (*b'*) **After death,** in the contents of the stomach or intestines; or owing to absorption, in the liver, kidneys, or other parts of the body. The longer the duration of the case, the less likely is it that any of the poison will be found after death in the contents of the stomach; and the more likely is it that if poison is detected at all in the body, it will only be found in some solid viscus. Hence the importance of submitting portions of these to analysis.

A poison having been found, it does not necessarily follow that the case is one of poisoning. Poison may be introduced into an article of food, in order to support a false charge. Again, poison may be introduced into evacuations, or even into viscera, with a similar object; or these may have become accidentally contaminated with poison from impurities in the containing vessel. Hence the importance of (1) if possible, securing for analysis vomit, &c., ejected in presence of the medical attendant; (2) using only thoroughly clean vessels for holding matters to be analysed, and (3) preserving such matters under seal, &c., so as to prevent their being tampered with. Suppose, however, that poison is found, and that such poison has not been introduced in one of the ways indicated above, the case may still not be one of poisoning, because the poison discovered—

(*a'*) **May be a natural constituent of articles of food,** *e.g.*, oxalic acid in combination is found in certain vegetables; or,

(*b'*) **May have been given in the course of medical treatment,** *e.g.*, arsenic or mercury (see these poisons).

In two other cases also, a poison, or substance resembling a poison, may be found in the viscera of an individual, and the case yet be not one of death from poison, viz.:—

(*c'*) **When death has been due to some other cause,** *e.g.*, drowning or hanging, operating before the poison has fully exerted its action on the system (see *ante*, p. 83); or,

(*d'*) **When the substance found is a "Ptomaine,"** or alkaloid resulting from decomposition (see *Ptomaines*, p. 308).

The total quantity of poison found in the viscera of an individual may be less than a minimum poisonous dose, and the case yet be one of death from poison. Frequently a large

proportion of the poison swallowed is got rid of by evacuation. In this way, the whole alimentary tract may be freed from the poison, and only that portion which has been absorbed remain in the body. This absorbed portion again is distributed more or less throughout the whole body. Obviously, however, only a fractional part of the body can be examined, and the quantity of poison found in this, therefore, is only a fraction of the quantity the body contains. Again, by elimination through the emunctories during life, a portion, or even the whole of the absorbed poison, may be removed from the body, and yet death occur from the effects of the poison. In such a case, the whole body may not contain such a quantity of the poison, as amounts to a minimum fatal dose. On the whole, therefore, the quantity of poison found in the body is, in the great majority of cases, of little importance. In a few cases, however, it may be important, *e.g.*, when the quantity found is small, and the poison is one sometimes present as a natural constituent of food, or sometimes given as a medicine. Hence, where possible, the quantity present should always be determined.

No poison having been found, the case may yet be one of poisoning—

(*a'*) **From the poison having disappeared** by evaporation, or having been removed from the body by evacuation or elimination. This, as already pointed out, is specially likely to occur in the case of very volatile, *e.g.*, gaseous, poisons; or in the case of very soluble poisons, *e.g.*, in poisoning by the corrosive acids; or in cases where an individual has lived for some time after swallowing the poison.

(*b'*) **From neglect to submit certain matters** (or a sufficient quantity thereof) **to analysis**, *e.g.*, in cases where the individual has lived for some time after administration of the poison, and no portion, or only very small portions, of the solid viscera, are submitted to the analyst. Again, of several articles of food, one alone may contain poison, and this may not have been submitted.

(*c'*) **From the poison having undergone chemical destruction** by oxidation or putrefaction. This may occur in the case of organic, but not in the case of inorganic poisons. It is possible that some organic poisons may undergo destruction, by oxidation in the body during life. Organic poisons, again, may be destroyed by putrefaction after death: some, however, *e.g.*, strychnia and opium, have been found to resist putrefaction for long periods.

(*d'*) **From there being no reliable means of extracting the poison** from substances containing it, or no satisfactory tests for its identification.

(*e'*) **From want of care or skill** on the part of the analyst.

Case B 3 (*R.* v. *Catherine Wilson*) is an example of a convic-

tion for murder by poison, notwithstanding the fact that no poison was discovered in the viscera of the persons poisoned.

Should a poison be found, a portion of it should, if possible, be preserved for production before the Court. (See Ind. Evid. Act, sect. 60, *ante*, p. 8.)

4. Experiments on Animals.

These may take the form of—

1. **Administration of suspected substances,** such as portions of—

(*a*) **Food.**—This is often employed as a rough preliminary test for the presence of poison.

(*b*) **Vomited matter.**—An experiment of this kind is sometimes the result of accident, and is open to the fallacy that morbid secretions, *e.g.*, bile, may, when swallowed by animals, cause symptoms of poisoning.

(*c*) **Eliminated poison.**—This is specially useful in the case of organic poisons for which there are no distinctive chemical tests, *e.g.*, aconitia and daturia (see, however, remarks on Ptomaines, p. 309). The term, a physiological test, is often used to denote a test for the recognition of a poison, dependent on its action on living animals.

2. **Comparison experiments.**—In a case of suspected poisoning by a substance, the action of which is not well known, it may prove useful to administer to an animal a dose of the poison supposed to have been employed, so that the symptoms present in the case may be compared with those which arise in the animal experimented on. Experiments of this kind are open to two objections.

(*a*) Some animals are apparently unaffected by poisons, which act violently on man, *e.g.*, pigeons appear to be unaffected by opium,[1] and some varieties of monkeys appear to be unaffected by strychnia.

(*b*) The symptoms produced in the animal experimented on may be different from those of the case, although the same poison was used in both; either from the action of the poison on the animal being different to its action on man, or from failure to properly proportion the dose to the size of the animal.

[1] See, however, p. 257.

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CHAPTER VIII.

IRRITANT POISONS CONSIDERED GENERALLY—CORROSIVE POISONS.

The general symptoms of irritant-poisoning are divisible into (*a*) Throat symptoms; (*b*) Abdominal symptoms; and (*c*) Final symptoms.

(*a*) **Throat symptoms.**—These are pain, difficulty in swallowing, and feeling of constriction; and (in corrosives) marks of corrosion in the mouth and throat.

(*b*) **Abdominal symptoms.**—These are epigastric pain, thirst, nausea, vomiting, purging, tenesmus, and dysuria. The stools and vomited matters often contain blood.

(*c*) **Final symptoms.**—These are inflammatory fever; or collapse accompanied by a quick feeble pulse, and cold sweats; sometimes the anus becomes excoriated. Various symptoms due to a special remote specific action of the poison, may also be present.

The order in which the symptoms appear varies according as to whether the case is one of corrosive, or of non-corrosive irritant poisoning. In corrosive poisoning, the throat symptoms appear first, and come on immediately, or almost immediately, and often the glottis and trachea are affected, causing dyspnœa. In non-corrosive irritant poisoning, the abdominal symptoms appear first, and are followed by throat symptoms. In non-corrosive irritants, the interval between swallowing the poison and first appearance of the symptoms varies; it may be very short in the case of the more soluble irritants, or may be half an hour or more in the case of less soluble ones.

Death in irritant-poisoning may occur—(*a*) Rapidly from **shock**, as in some cases of arsenical poisoning; or from **suffocation**, as in some cases of corrosive poisoning. (*b*) Less rapidly from **syncope**, due to absorption and secondary action, as in some cases of oxalic acid poisoning. (*c*) Still less rapidly from **exhaustion**, due to protracted irritation; or, (*d*) In corrosive poisoning, after a considerable period, from **starvation** or **suffocation**, the result of local injury.

The general post-mortem appearances of irritant-poisoning, are signs of irritation or corrosion of the mucous membrane of

the alimentary canal. In some cases, similar signs may be present in other situations (see *ante*, post-mortem appearances, p. 123).

As regards the treatment in cases of irritant-poisoning, the following points may be noted :—

(*a*) **Elimination.**—Usually there is free vomiting, which should be encouraged by copious draughts of warm water. In some cases, emetics or the stomach-pump may be required; the latter, however, should never be used in corrosive-poisoning.

(*b*) **Prevention of action.**—The means whereby this indication may be carried out have already been sufficiently indicated (see *ante*, p. 118). Here it may be noted that, in the case of vegetable and animal irritants, antidotes are, as a rule, not available.

(*c*) **Counteraction and removal of effects.**—Under this head the employment of measures calculated to allay irritation —among them administration of demulcents—is indicated. Oily demulcents must not be given in poisoning by phosphorus, or by cantharides, these poisons being soluble in oil. Stimulants may be given to counteract depression. In cases of corrosive-poisoning, laryngotomy may be required (see Mineral Acid Corrosives, p. 131).

Irritant poisons may be conveniently classified as follows:— I. Corrosive poisons. II. Organic acid and non-metallic irritants. III. Metallic irritants. IV. Vegetable irritants. V. Animal irritants; and, VI. Mechanical irritants.

IRRITANTS, CLASS I.—CORROSIVE POISONS.

Sub-Class I.—Mineral Acid Corrosives.

The principal poisons of this sub-class are :—

(1) **Sulphuric acid**, or oil of vitriol (H_2SO_4).
(2) **Hydrochloric** or **muriatic acid**, or spirit of salt (HCl).
(3) **Nitric acid**, or aqua fortis (HNO_3).

Action, and Origin of Cases.—These three acids are very similar in action, and are powerful corrosives, except when much diluted, when they act as simple irritants. Cases of poisoning by them are rare in India,[1] but tolerably frequent in Europe. Owing to their marked properties, these acids are **seldom used homicidally**; a few cases, however, of homicidal poisoning of children by sulphuric acid, are recorded. **Accidental cases**, except among children, also are rare. Most commonly adult cases of poisoning by these acids are **suicidal**, and in England form about one-twelfth of the total suicides by poison. Sulphuric acid has been injected by mistake into the rectum as an enema,

[1] I have met with only one case (suicide by nitric acid) in Bombay in twenty years.

and has been thrown up into the vagina, for the purpose of procuring abortion. Not infrequently in England, and in rare cases also in India, sulphuric acid is thrown over the person in order to cause injury (vitriol throwing). Sometimes nitric acid is used in the same way. A few accidental fatal cases from inhalation of the vapours given off by nitric acid (see *Nitrous Acid*, p. 141) have occurred; and a case of homicide by pouring nitric acid into the ear during sleep, is on record.

General symptoms.—Swallowed in a tolerably concentrated condition, these acids cause: Immediate burning pain in the mouth and throat, followed by pain in the abdomen. Vomiting of brown or black matter containing blood, mucus, and shreds of mucous membrane. The vomited matters, especially those first ejected, may effervesce on coming into contact with the ground (owing to the acid acting on carbonates). There is tenesmus but no purging; difficulty and pain in micturating, in swallowing, and often also in breathing. The lips and interior of the mouth, unless the poison has been conveyed to the back of the throat by a spoon or some such means, are discoloured, or shrivelled and blistered. The discoloration, at first white, afterwards becomes ash grey or brown, or, if nitric acid has been employed, turns yellow. Marks of the action of the acid may be found on the skin or clothes; these are yellow if from nitric acid, and brown —or, if on coloured cloth, dull red—when due to sulphuric acid. Hydrochloric acid does not stain the skin, but stains coloured cloth very much like sulphuric acid.

Special symptoms and exceptional cases: (1) **Sulphuric acid.**—Salivation coming on about the second or third day, has been observed in several cases. In exceptional suicidal cases there has been considerable delay in the appearance of serious symptoms (see *Cases* B 7 and 8), and in one case vomiting ceased in four hours, and did not return, though the patient lived thirty-one hours. In a few cases sulphate of indigo—a solution of indigo in strong sulphuric acid, used in dyeing—has been taken, giving rise to symptoms exactly like those of sulphuric acid poisoning; except that the mouth and vomited matters, and in some cases the urine also, are tinged blue. (2) **Hydrochloric acid.**—In one case of poisoning by this acid salivation came on rapidly, in another convulsions preceded death, and in a third delirium came on on the second day, followed by paralysis of the limbs. The vapour of hydrochloric acid if inhaled acts as a poison, causing great irritation of the air passages. (3) **Nitric acid.**—In one case of poisoning by this acid lock-jaw was present, and in another insensibility. A case is recorded of poisoning by a mixture of nitric and sulphuric acids. **Nitro-muriatic acid** is used in the arts for dissolving gold and other purposes, but does not seem to have given rise to any cases of poisoning.

Mode and time of death.—Death may occur rapidly from shock or suffocation. Children poisoned by sulphuric acid often die from the latter cause, the poison never reaching the stomach. Hydrochloric acid poisoning is apt also to end in death by suffocation. Death may take place less rapidly from exhaustion; or, after months, from starvation, due to stricture of the œsophagus. In the case before referred to, where nitric acid was poured into the ear, death took place in thirteen weeks, from necrosis and inflammation spreading to the brain. In cases of recovery from the first effects, severe dyspepsia is often observed, and may continue throughout life. Death usually takes place within twenty-four hours, but has occurred (in nitric acid poisoning) in an infant in five minutes, and in an adult in one hour and three-quarters. The longest fatal periods recorded are, in sulphuric acid poisoning forty-five weeks, and in nitric acid poisoning two years, both from starvation.

Fatal dose.—The more concentrated the form in which these acids are swallowed the more likely is a given quantity to cause death. A very few drops of any of the three acids may cause death from suffocation; and the more empty the stomach, the more likely is serious injury to it to result. Hence the least quantity required to destroy life cannot be precisely stated. The smallest doses which are recorded to have proved fatal are sulphuric acid, 1 drachm; nitric acid (in a child of thirteen), 2 drachms; and hydrochloric acid about ½ ounce. The largest non-fatal dose of sulphuric acid recorded is 3 ounces; and several instances of recovery after swallowing an ounce of hydrochloric acid are reported.

Post-mortem appearances.—These are usually: marks of the acid, as before described, on the clothes, skin and lips, and in the mouth. Signs of inflammation and corrosion in the œsophagus, and sometimes in the larynx. Stomach in the majority of cases discoloured (yellow from nitric acid, brown or black from the other two acids), inflamed, corroded, and sometimes perforated. Marks of the action of the acid may be absent from the mouth, if the acid has been poured down the throat with a spoon; absent, or nearly absent, from the œsophagus, even although the poison has reached the stomach; and even in fatal cases, altogether absent from the stomach. **Perforation of the stomach** has been found in about one-third of the fatal cases of sulphuric acid poisoning, is rare in nitric acid, and still rarer in hydrochloric acid poisoning.

Treatment.—The stomach-pump must **not** be used. Give calcined magnesia, carbonate of magnesia, chalk, or carbonate of soda, followed by mucilaginous drinks. If death from suffocation threatens, laryngotomy must be performed. In the after treatment leeches and other antiphlogistic remedies may be required. Excoriations should be washed with lime water, and treated as

burns. Distress due to inhalation of hydrochloric acid vapour, may be relieved by inhalation of weak ammonia.

Detection.—In fatal cases of poisoning by these acids, especially if life has been prolonged for two or three days, no trace of the poison may be discoverable in the viscera. Should the presence of one of these acids be detected, it is important—salts of these acids being common constituents of food and medicine—to ascertain whether any of it is present in the free condition. If no free acid be found, the quantity of **combined** acid present becomes of importance. The quantity of **free** acid present is specially important in hydrochloric acid poisoning, as this acid (in loose combination with pepsin), is contained uncombined with bases, in the gastric juice, to the extent of about ·2 per cent. or more.

Sulphuric acid and solutions of sulphates give a white precipitate with barium nitrate, which is (1) insoluble in dilute nitric acid, (2) insoluble in water, and (3) when collected, dried, and heated with powdered charcoal before the blowpipe, converted into barium sulphide, soluble in hydrochloric acid with escape of hydrogen sulphide, recognised by its odour, and by its blackening paper wetted with lead acetate solution.

Free sulphuric acid chars organic matter. It may be separated from soluble sulphates by concentration on a water bath and treatment with quinine; separating the quinine sulphate formed, after thorough drying, by strong alcohol, in which quinine sulphate is soluble, but alkaline and metallic sulphates are insoluble. The alcoholic solution is then to be evaporated to dryness, the residue dissolved in boiling water, decomposed by ammonia, filtered, and the sulphuric acid estimated in the filtrate by precipitation as barium sulphate.

Hydrochloric acid and solutions of chlorides give (1) a white flocculent precipitate with silver nitrate solution, soluble in ammonia, but insoluble in boiling nitric acid, and (2) when boiled with H_2SO_4 and manganese dioxide, evolve chlorine, recognisable by its colour, odour, and bleaching action on moistened litmus paper.

Free hydrochloric acid evolves chlorine when boiled with manganese dioxide only, and (2) when mixed with HNO_3 dissolves gold. Organic mixtures to be tested for the free acid should be distilled, and the distillate tested for HCl; or if this, as sometimes happens, fails, resort may be had to either (1) the quinine process as for sulphuric acid described above, estimating the chlorine in the decomposed filtrate volumetrically with silver nitrate solution; or (2) the organic mixture may be divided into two equal portions, and one of these neutralised by sodic carbonate; both are then evaporated to dryness, the residues incinerated, and the chlorine in each separately estimated. The excess of chlorine in the neutralised portion, corresponds to the free acid present in the original fluid.

Nitric acid and solutions of nitrates (1) heated with H_2SO_4 and fragments of copper dissolve the copper with escape of lower oxides of nitrogen, known by their red colour and their liberating iodine from potassium iodide; (2) boiled with H_2SO_4 and a drop or two of indigo solution, decolorise the indigo (this test by

itself is not conclusive evidence of the presence of HNO_3); and (3) if to a portion of the solution under test, ferrous sulphate solution and then a little H_2SO_4 be cautiously added, a brown ring appears at the point of contact of the H_2SO_4 with the other fluids.

Free nitric acid gives the above reactions without the addition of H_2SO_4, and if mixed with HCl dissolves gold. It may be separated from organic mixtures by the quinine process described above for sulphuric and hydrochloric acids.

Stains on Cloth, &c.—The yellow stains of nitric acid on the tissues or on cloth, treated with weak caustic potash solution, acquire an orange colour, while iodine stains disappear, and bile stains remain unaltered. Stains of sulphuric or hydrochloric acid on dark-coloured cloth, are usually reddish, the red tint disappearing on addition of ammonia. Sulphuric acid stains are more moist, and show more evidence of corrosion, than hydrochloric acid stains. Stains on cloth, &c., should be macerated in water which will acquire an acid reaction if free acid is present in the stain. The watery solution may then be tested for the suspected acid. A comparison experiment should at the same time be made with an unstained portion of the cloth. Burns must not be mistaken for marks of corrosion by sulphuric acid. Blyth, on the authority of Maschka, mentions a case, where free sulphuric acid found in a charred mark on an infant's bed, was ascertained to be due to the sudden quenching with water of a live coal which had fallen thereon.

The detection of these acids may be required in criminal cases other than cases of poisoning or causing hurt. I have been called upon to identify sulphuric acid, in a case where a bottle of this liquid, loosely stoppered and leaking, was sent by post, and a prosecution under the Post Office Act thereupon instituted against the sender. A mixture of HNO_3 and H_2SO_4 is used in making nitro-glycerine and other explosives; the identification of these acids, therefore, might be required in support of a charge of illicitly manufacturing such substances. Nitric acid is used in India for the purpose of "sweating" silver coin; the method employed being apparently to steep the coins for a short time in this acid, and then, by adding metallic copper, precipitate and recover the silver. Hydrochloric acid has been used in forgery, to remove marks of writing ink from paper. Hydrochloric acid gas acts injuriously on vegetation, and by law in England alkali manufacturers—making carbonate of soda by the salt cake process—render themselves liable to penalties if they fail to condense to a stated extent, the hydrochloric acid evolved in the manufacture.

Hydrofluoric acid, HF, used for etching on glass, is a powerful corrosive. One fatal case is recorded of poisoning by this acid, in which half an ounce was swallowed, and death took place in thirty-five minutes.

Corrosives, Sub-Class II.—Alkaline Corrosives.

The chief poisons of this sub-class are the caustic alkalies: **potash, soda, and ammonia; and their carbonates.** These, like the acid corrosives, act as simple irritants when sufficiently diluted. Cases of poisoning by the alkaline corrosives are rare. I have met with only one fatal case in Bombay in twenty years, viz., a case of suicide by caustic ammonia. In Europe, cases of poisoning by the corrosive alkalies are commonly accidental, and owe their origin to the extensive use of these substances—carbonates of potash and soda especially—in the arts. Impure carbonate of soda is sold in the bazaars of Bombay under the names of **Sajjikhara** and **Bangadakhara**; impure carbonate of potash, as **Javakhara**, and the mixed carbonates as **Papadakhara.**[1]

General symptoms.—These are similar to those caused by the corrosive acid, except that the vomited matters are alkaline, and do not effervesce on the ground, and purging—not present in poisoning by the corrosive acids—is a frequent symptom in alkaline poisoning. In poisoning by ammonia or its vapour, and by the carbonates of ammonia, inflammation of the air-passages is a constant symptom. Caustic ammonia is less powerfully corrosive than caustic potash and caustic soda, and carbonate of soda is less corrosive than carbonate of potash. As in poisoning by the corrosive acids, the greater the degree of concentration of the poison, the greater the danger.

The post-mortem appearances and modes of death are similar to those in poisoning by the corrosive acids. **The treatment** also is the same, except of course, that dilute acids, preferably dilute vegetable acids, should be given, instead of dilute alkaline solutions. In cases of poisoning by ammonia, inhalation of acetic acid vapour may be used, to allay irritation of the air-passages.

Fatal dose, &c.—Forty grains of caustic potash caused the death of an adult in seven weeks, from exhaustion. About half an ounce may be looked on as an ordinary fatal dose of caustic potash or caustic soda. About half an ounce of carbonate of potash has in more than one instance, in adults, caused death in two to four months. Carbonate of soda is much less poisonous: a case of recovery after swallowing twelve ounces is on record. As regards caustic ammonia, a quarter of an ounce of the strong solution has caused death, and half an ounce may be regarded as an ordinary fatal dose.

Liquor potassæ (B.P.) contains 5·84, and liquor sodæ (B.P.) 4·1 per cent. of caustic alkali. Strong solution of ammonia (B.P.) contains 32½ per cent., and solution of ammonia (B.P.)

[1] Sakharam Arjun, Catalogue of Bombay drugs.

10 per cent. of NH_3. Compound camphor liniment (B.P.) contains about 7·3 per cent. of NH_3,[1] and has given rise to more than one case of poisoning.

Detection.—Free potash and soda are most conveniently separated from organic mixtures by dialysis; after which the quantity present may be estimated (in the fluid which has passed through the membrane) by a standard acid. Free ammonia is best separated by distillation. Potash and ammonia are distinguished from soda by giving, (1) a precipitate with tartaric acid in excess, and (2) a precipitate with platinic chloride in presence of hydrochloric acid. Ammonium may be distinguished from potassium salts, (1) by their volatility, and (2) by their evolving ammonia when heated with solution of caustic potash.

Lime, CaO.—May be included among the alkaline corrosives, but from its little solubility, is much less dangerous than the poisons just described. The symptoms, treatment, &c., are the same as in poisoning by potash and soda. Malingerers have been known to produce ophthalmia, by applying lime to the conjunctiva.

Other corrosives.—Certain metallic salts, *e.g.*, mercuric chloride and zinc chloride, possess a corrosive action: these will be described with the other compounds of these metals. A more or less destructive action on the tissues is also exerted by oxalic and acetic acid, see vegetable acids; by bromine, see non-metallic irritants; and by carbolic acid, see narcotics, group 2.

[1] The corresponding I.P. preparations are of the same strength.

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CHAPTER IX.

IRRITANTS, CLASS II.—ORGANIC ACID AND NON-METALLIC IRRITANTS.

Sub-Class I.—Organic Acids.

Oxalic Acid ($H_2C_2O_4 2H_2O$).

POISONING by oxalic acid is rare in India, but tolerably frequent in Europe. In England, in the five years ending 1880, seventy-four deaths from oxalic acid were registered, of which sixty-five (about seven-eighths) were suicidal, and the rest accidental. I have met with but four fatal cases in Bombay in twenty years. Of these three were suicidal, and the fourth apparently homicidal. Many accidental cases owe their origin to the resemblance in appearance of oxalic acid to sulphate of magnesia. Homicidal cases (probably owing to the strongly acid taste of the poison) are rare. Oxalic acid and the acid alkaline oxalates are chiefly used in the arts for cleansing purposes, *e.g.* cleansing leather, wooden boards, &c., and removing ink stains and iron moulds from linen.

Symptoms.—Oxalic acid (and the acid alkaline oxalates) possess both a local and a remote action. Of these, the remote action is much the more serious, and is usually the cause of death. **Local action.**—This is corrosive or irritant in character according to the degree of concentration of the poison. Swallowed in concentrated solution, the symptoms due to the local action of the poison are immediate burning pain in the mouth and throat, with sense of constriction, followed rapidly by pain in the abdomen, and vomiting of matters containing altered blood. The interior of the mouth has a bleached white appearance; and if the patient lives long enough, purging sets in, the stools containing blood. Swallowed in dilute solution, the symptoms due to local action are those of non-corrosive irritant-poisoning. There is an acid taste, but no burning pain, in the mouth; and vomiting does not come on for fifteen or twenty minutes: in one case it did not come on for seven hours. **Remote action.**—The symptoms due to this are twitchings of the muscles, in some cases amounting to tetanic convulsions; numbness, tingling and cramp in the limbs; great depression of the heart's action;

slow spasmodic respiration; collapse, and stupor or insensibility, and sometimes delirium. These symptoms may be unaccompanied, or almost unaccompanied, by vomiting, pain, and other symptoms of local action. **After symptoms.**—In cases of recovery, loss of voice has been observed, in one case complete for eight days, in another partial for more than a month. Alteration of the voice is sometimes present in acute cases. Numbness and tingling of the limbs and twitchings of the muscles, may remain for some time after the first effects of the poison have disappeared. So also may irritability of the stomach; and as in other cases of corrosive poisoning, death may occur after a considerable interval, from starvation.

Fatal period.—Exceptionally short, usually under one hour. Death has occurred in three minutes, in ten minutes; and in one case, not until the fourteenth day, from starvation. Woodman and Tidy mention a case where a man is reported to have walked ten miles after swallowing an ounce of oxalic acid.

Fatal dose, about half an ounce; cases, however, are reported of death from one drachm (in a boy æt. 16), and three drachms (in a female æt. 28), death taking place in respectively twenty-one hours and one hour. Cases of recovery after swallowing an ounce or more, are reported.

Post-mortem appearances.—These vary according to the degree of concentration of the poison and rapidity of death. If the poison has been swallowed in a tolerably concentrated form, the lining membrane of the mouth, throat, and gullet is found white, shrivelled, and easily detached. If death has been rapid, the mucous membrane of the stomach may be pale, but usually is deep red, in places black, and may be found eroded. Perforation is rare. The stomach has been found so soft as to tear easily. The intestines may be found inflamed, and the lungs are often congested. Congestion of the brain has been found; and in one case, probably from violent vomiting, apoplectic effusion was present.

Treatment.—Administration of saccharated solution of lime, or of chalk suspended in water. Magnesia or carbonate of magnesia may be given instead of chalk. After administration of antidotes, warm water may be given freely. Vomiting should be promoted. Alkalies are inadmissible, and the stomach-pump should not be used if much corrosion be present, and if used should be introduced with great caution.

Acid alkaline oxalates.—Two of these are in common use in the arts, viz. binoxalate of potash, $KHC_2O_4\,H_2O$, and quadroxalate of potash, $KHC_2O_4\,H_2C_2O_4\,2H_2O$. Both are sold under the names of salt of sorrel, and essential salt of lemons, and both are nearly as poisonous as oxalic acid. The binoxalate has caused death in eight minutes. Half an ounce of it has proved fatal, but recovery is recorded from one ounce. The

symptoms, &c., are precisely the same as in poisoning by oxalic acid.

Detection.—It should be remembered that alkaline oxalates are found in many plants, *e.g.* in wood sorrel, and in its Indian substitute Rumex vesicarius (*Chuka*); and also that oxalate of lime is found in many plants.[1] In cases, therefore, of alleged poisoning by oxalic acid or an oxalate, the post-mortem appearances are, and the determination of the quantity of poison may be, of great importance. Oxalic acid is entirely dissipated by heat. In solution it yields: (1) A white precipitate with $AgNO_3$, insoluble in acetic acid, but slowly soluble in cold HNO_3, and which, when collected, dried, and heated, is converted into metallic silver with a slight explosion. (2) A white precipitate with sulphate of lime solution insoluble in acetic acid, which, when dried and heated, becomes converted into carbonate of lime without charring. **From organic mixtures,** oxalic acid and soluble oxalates may be separated by boiling with water, filtering, and precipitating the filtrate with acetate of lead; subsequently decomposing the oxalate of lead by suspending it in water, and subjecting it to the action of a current of hydrogen sulphide. Insoluble oxalates should be first decomposed by boiling with solution of caustic potash.

Acetic acid, $HC_2H_3O_2$; Tartaric acid, $H_2C_4H_4O_6$; and Citric acid, $H_3C_6H_5O_7, H_2O$.

Acetic acid acts as a corrosive if concentrated, but as an irritant when dilute; one fatal case is recorded in a girl æt. nineteen, and another in a child æt. two. Vinegar contains about 5 per cent. of this acid, and might possibly, in large doses, act as a poison. Acetic acid may be separated from organic mixtures by distillation and recognised (1) by its odour, and (2) by boiling with sulphuric acid and alcohol, when a peculiar aromatic smelling vapour (acetic ether) is evolved. **Tartaric acid and Citric acid** in large doses act as irritant poisons; one ounce of tartaric acid has caused the death of an adult, and one fatal case also in an adult, from four or five tablespoonfuls of cream of tartar (potassium bitartrate) is on record. Citric acid is believed to be more poisonous than tartaric acid.

Sub-Class II.—Non-Metallic Irritants.

Phosphorus.

Red or amorphous phosphorus is not poisonous. Ordinary yellow phosphorus, especially in fine division, is an extremely active compound irritant poison. Yellow phosphorus in fine division is contained, to the extent of from about 1½ to 4 per

[1] *E.g.*, in rhubarb root and squills. See also Arums, p. 212.

cent., in various pastes used for destroying vermin, and to a varying extent, usually about 15 per cent., in the composition with which the heads of some kinds of lucifer matches are tipped.[1] Cases of poisoning by solid phosphorus usually arise from swallowing vermin pastes, or lucifer match-heads, and are generally in adults suicidal, and in children accidental. Phosphorus vapour is also highly poisonous, but the symptoms produced by it usually differ from those of poisoning by solid phosphorus (see below).

In poisoning by solid phosphorus (acute poisoning).—The symptoms may appear almost immediately, but in many cases do not appear for one to six hours; in a few cases their appearance has been delayed longer, and one case is recorded where five days elapsed before they appeared. The first symptoms are those of ordinary irritant poisoning with the following points of difference: (1) the breath may be phosphorescent and have a garlicky odour; (2) the vomited matters and other evacuations may be phosphorescent; and (3) diarrhœa is sometimes absent. Subsequently jaundice sets in, usually before the end of the third day, often after a remission of the symptoms, and is accompanied most commonly by (*a*) retention of urine followed by fatal coma, delirium being sometimes present; or less commonly by (*b*) hæmorrhage from the mouth, bowels, and genito-urinary organs, and spots of purpura under the skin; with death ultimately from exhaustion; or still more rarely by (*c*) cramps and fatal tetanic convulsions. Death in a few cases occurs before the end of the second day and before jaundice has set in. Usually death takes place within a week. In one case the patient survived eight months. A little over one-ninth of a grain has caused death. Three-quarters of a grain to two grains may be looked on as an ordinarily fatal dose; recovery, however, has been recorded after swallowing five grains.

Phosphorus vapour.—One or two acute cases of poisoning by phosphorus vapour are on record, but as a rule this form of poisoning is chronic in character. The chief symptoms present in chronic cases are, caries of the teeth, and painful necrosis of the jaws, most commonly of the lower jaw, followed in many cases by death from debility. Cases of this form of poisoning have chiefly been observed in workers in phosphorus, especially lucifer match makers: hence the term "lucifer match maker's disease," applied to this form of poisoning. Owing to improvements in the method of manufacture of lucifer matches, and specially to the introduction of red as a substitute for yellow phosphorus, cases of this form of poisoning are now much less frequent than formerly.

[1] In other kinds, the non-poisonous red phosphorus is substituted for the poisonous yellow variety; and the heads of "safety" matches contain no phosphorus.

Post-mortem appearances.—In acute cases fatty degeneration of the liver is always, or almost always, present, and has been found far advanced in a case where death occurred in twenty-four hours. The phosphorus liver, except that as a rule it is enlarged and not diminished in size, resembles to the naked eye the liver of acute yellow atrophy. Fatty degeneration also may be present, of the muscular fibres of the heart, of the kidneys, and of the epithelial cells of the intestinal mucous membrane. Spots of extravasation are often present under the serous and mucous membranes, and in other tissues. The skin may be yellow and the stomach contents phosphorescent. Signs of inflammation of the mucous membrane of the alimentary canal are not commonly present. In exceptional cases the post-mortem appearances have been entirely negative.

Treatment.—In acute cases, emetics may be given and vomiting promoted, or the stomach-pump used. No oil or fatty matters should be given, as these dissolve phosphorus. Turpentine in 40-minim doses often repeated, is recommended as an antidote.

Detection.—Phosphorus readily undergoes oxidation in the body, hence after death analysis may fail to detect its presence. Solid yellow phosphorus is easily recognised by its physical characters. If present in organic mixtures in considerable quantity, it may be separated as a sediment by washing, afterwards melting it, if finely divided, under warm water; or it may be extracted from organic matters by carbon disulphide, in which it is soluble. If present only in small quantity, one of three processes may be employed, viz. (1) Mitscherlich's. Acidulate the matters with H_2SO_4 and distil them to dryness in the dark, using a well-cooled glass condensing-tube, which will show luminosity if phosphorus be present. (2) A modification of Marsh's process, by which phosphorus, if present, becomes converted into gaseous hydrogen phosphide (PH_3), which burns with an emerald green flame, and produces a black precipitate in silver nitrate solution, the liquid, after removal of the excess of silver, showing the presence of free phosphoric acid. (3) Lipowitz's method. Acidulate the matters with H_2SO_4, and boil fragments of sulphur in them for an hour; remove and wash the fragments of sulphur, which, if phosphorus is present, will be found to have become luminous in the dark, owing to the deposition of phosphorus upon them. Phosphorus boiled with HNO_3 becomes converted into phosphoric acid, the presence of which may be recognised by precipitation with a mixture of ammonia, ammonium chloride, and magnesium sulphate solutions. In this way, also, its quantity may be estimated.

Chlorine, Bromine, and Iodine.

Chlorine, largely used as a disinfectant and in bleaching, is a highly irritant gas, causing great irritation and inflammation of the air-passages, and acting as an irritant also on the conjunctiva. Workmen in chlorine factories are, however, said to become in time to a certain extent habituated to its presence. **The hypochlorites** contained in bleaching-compounds, *e.g.*, chloride of lime and liquor sodæ chlorinatæ, also act as irritant poisons.

Treatment.—Inhalation of diluted hydrogen sulphide; this, however, must be employed with caution, as hydrogen sulphide is in itself highly poisonous.

Bromine.—This in the liquid form is a corrosive poison. In the form of vapour its effects are similar to those produced by chlorine. One case of death in seven hours from an ounce of liquid bromine is recorded. **Bromide of potassium** in single doses has not been known to give rise to symptoms of poisoning. In large doses taken for a considerable time, it causes impairment of the functions of the brain and spinal cord, such as diminished activity of reflex action, cloudiness of intellect, impairment of memory and of articulation, and tendency to stupor.

Iodine.—Free iodine is a corrosive irritant; in poisoning by it the vomited matters are often blue or black owing to its action on starch. Twenty grains has caused death, but recovery is recorded from 1½ drachms. Iodine in small doses, often repeated, is liable to give rise to chronic poisoning, the chief symptoms of which are, irritability of the stomach, vomiting and purging, accompanied by salivation and wasting of the body generally, and specially of the breasts or testicles. **Iodide of potassium** has frequently been given medicinally in comparatively large doses (100 grains or more a day) without producing ill-effects. Sometimes, however, small doses, often repeated, give rise to symptoms resembling those of severe catarrh, and in exceptional cases such symptoms have even arisen from single small doses. In exceptional cases also, the ordinary symptoms of catarrh have been accompanied by somewhat severe symptoms of irritant poisoning.

Nitrous Acid.

The vapours of nitric acid, and the red gas evolved during the oxidation of matters by nitric acid,[1] are highly poisonous, giving rise when inhaled to inflammation of the air-passages and lungs. A few cases of death from the inhalation of such vapours are on record. In some of these the fatal result has followed on ex-

[1] Nitrogen peroxide (NO_2) and nitrous anhydride (N_2O_3); the latter, by combination with water, yields nitrous acid (HNO_2).

posure for a few minutes to the vapours arising from a quantity of nitric acid, accidentally spilled by the breaking of a large vessel filled therewith. In these cases a noticeable feature has been the slight amount of discomfort felt for the first two or three hours after the accident, death nevertheless occurring rapidly (within ten to fifteen hours).

Sulphur dioxide, SO_2, commonly called Sulphurous Acid.

This gas, like nitrous acid, acts as an irritant to the air-passages. From Woodman and Tidy's experiments it appears that when the quantity is small, animals soon get accustomed to its presence, and thereafter do not suffer nearly so much from its irritant action.

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CHAPTER X.

IRRITANTS, CLASS III.—METALLIC IRRITANTS.

Sub-class I.—Volatile Metals.

ARSENIC, As = 75.

COMPOUNDS of this metal act (*a*) as irritants, and (*b*) remotely on the nervous system; hence in cases of arsenical poisoning there may be present: 1. Irritant symptoms; and 2. Nervous symptoms.

1. **Irritant symptoms.**—Arsenical compounds act as irritants to the mucous membrane of the stomach and intestines, exerting this action even when introduced into the system by channels other than the mouth, *e.g.*, even when absorbed through a wound. They also exert an irritant action on the various emunctories. Hence in cases of arsenical poisoning there may be present: (*a*) The usual symptoms of irritant poisoning (unaccompanied, however, by any metallic or acid taste in the mouth); viz., epigastric and abdominal pain, thirst, vomiting, tenesmus, purging, &c., with presence of blood in the vomit and stools; and (*b*) Symptoms due to the irritant action of the poison on the skin, kidneys, liver, &c., such as conjunctivitis, painful cutaneous eruptions and desquamation of the cuticle, salivation, dysuria, and even suppression of urine leading to uræmic coma, and jaundice.

2. **Nervous symptoms.**—The symptoms resulting from the remote action of arsenical compounds on the nervous system vary greatly in different cases. Thus there may be: (*a'*) **Collapse**, with coldness of the surface and feeble pulse; or (*b'*) **Numbness and tingling** of the extremities, cramps, and even paralysis; or (*c'*) **Convulsions**, choreic, epileptiform, or tetanic in character, and lock-jaw; or (*d'*) **Delirium**, and even acute mania; or (*e'*) **Headache**, drowsiness, and stupor, deepening into coma.

Types of arsenical poisoning.—In the great majority of **acute cases**, well-marked and severe irritant symptoms are present. Such cases either (*a'*) prove rapidly fatal—say within twenty-four hours—by collapse; or (*b'*) the usual symptoms of irritant poisoning are followed by various nervous symptoms, the patient either recovering; or dying in one or other of a variety of modes, *e.g.*,

from exhaustion, or by coma, or in tetanic convulsions. In some of these compound irritant cases, remissions, or even intermissions, of the irritant symptoms, have been observed. In a few acute cases, irritant symptoms are absent, or slight, and the nervous symptoms well marked (see *Cases* B 9); such cases usually prove rapidly fatal, either by collapse or by coma. **In chronic cases** both irritant and nervous symptoms are usually present Sometimes in chronic cases the amount of gastric irritation is slight, while the irritant action of the poison on the skin, and emunctories other than the intestines, is well marked. In chronic cases, the nervous symptoms frequently take the form of numbness and tingling of the extremities, and paralysis.

Diagnosis from disease.—**Acute** irritant cases with collapse greatly resemble cholera; and in India a common way of attempting to conceal homicidal poisoning by arsenic, is to report the case as one of death from this disease. Sometimes, also, especially in cases where two or more persons, after partaking of food in company, are attacked by cholera in quick succession, a groundless suspicion of arsenical poisoning arises. The chief points which distinguish arsenical poisoning from cholera, are: 1. The presence in the former of blood in the stools; 2. The absence in the former of the rice-water appearance of the stools, characteristic of cholera (this appearance may, however, be present in the later stages of arsenical poisoning), and, 3. In cholera, pain in the throat does not precede vomiting; while in irritant poisoning the reverse is the case. The prevalence or absence of cholera in the locality at the time may also serve as an aid to the diagnosis. Cases where the irritant symptoms are slight, are sometimes not recognised during life as cases of poisoning. **In chronic cases,** persistent gastric irritation not yielding to treatment, accompanied by numbness and tingling of the extremities, with tendency to paralysis, should arouse suspicion, and indicate the necessity of subjecting to analysis, the urine or other evacuations.

Interval between swallowing the poison and first appearance of symptoms.—This is usually half an hour to an hour. Cases, however, have been reported where the symptoms appeared almost immediately. Taylor[1] mentions a case where the symptoms came on while a man was in the act of eating a cake containing the poison. I once met with a case where the symptoms appeared while a man was drinking a cup of tea, made with water from a kettle, into which arsenious oxide had been introduced. On the other hand, a few cases are reported in which this interval has been two, to eight or nine hours. In some of these cases the prolongation of the interval is difficult to account for; in others it appears to have been due to fulness of the stomach, to sleep, or to intoxication (see *Cases* B 10).

[1] Taylor on Poisons, p. 292.

Fatal period.—In acute cases this is usually under twenty-four hours. In many cases, especially those in which marked nervous symptoms appear early, death takes place in under twelve to fourteen hours (see *Cases* B 9). In one case a young man died with tetanic symptoms in twenty minutes;[1] this is the shortest fatal period recorded. Longer fatal periods than three days are sometimes met with. Taylor[2] mentions cases of death in six days, seven days, fifteen days, and sixteen days. In one case (a woman accidentally poisoned by external application of a solution of arsenic), death did not occur for two years.

Post-mortem appearances.—Usually the **gastric mucous membrane** is reddened from inflammatory action, and has been found so even where the poison has been introduced, by channels other than the mouth. It has been found intensely inflamed even when death has taken place within two hours after swallowing the poison. The gastric mucous membrane may be reddened in patches, or the redness may have a dotted or striated appearance, or the whole mucous membrane may be deep red, with dark spots or patches from underlying extravasated blood. Frequently the inner surface of the stomach has a corrugated appearance, and is covered with tenacious mucus entangling particles of the poison. Its contents are often dark in colour from altered blood. Sometimes yellow patches are found (due to conversion of the arsenic into sulphide), on the mucous membrane of the stomach or intestines. The redness sometimes extends into the **duodenum**; more rarely the intestines are found inflamed throughout their whole length. Commonly the rectum is found inflamed. **Ulceration** of the gastric mucous membrane is not common, but has been found in a case of death in ten hours. **Perforation** of the stomach is very rare, but is occasionally found (see *Cases* B 11). Sometimes in fatal cases the stomach and intestines show little or no signs of inflammatory action; this has been observed even in a case where well-marked irritant symptoms were present during life (see *Cases* B 12). Congestion, or lividity of the **endocardium**, especially of the left ventricle, is frequently—according to some almost invariably—found. Harvey[3] mentions that, out of thirty-three cases in which the condition of the heart was noted, in eight only was the endocardium found natural. Congestion of the brain, lungs, kidneys, or liver is sometimes met with (see *Case* B 11 *b*). Arsenic exerts a marked antiseptic action on the tissues, and hence, in fatal cases, post-mortem appearances of gastric irritation may remain recognisable for a considerable period. Post-mortem appearances indicative of death from arsenical poisoning, have been found coupled with post-mortem appearances pointing to death, from mechanical violence (see *Cases* A 18, *a* and *b*); hanging (see *Case* A 27 and

[1] Taylor on Poisons, p 308. [2] *Ibid.*, pp. 27, 309.
[3] Bengal Medico-legal Report for 1870-72, p. 255.

p. 83); and in bodies found under circumstances pointing to death by drowning (see *Case* A 18 *c*).

Mortality.—Guy states this at 52 per cent. The reports furnished to the Bombay Chemical Analyser, for the ten years ending 1884, show a total of 652 persons poisoned, and 305 deaths, equal to a mortality of about 45¼ per cent.

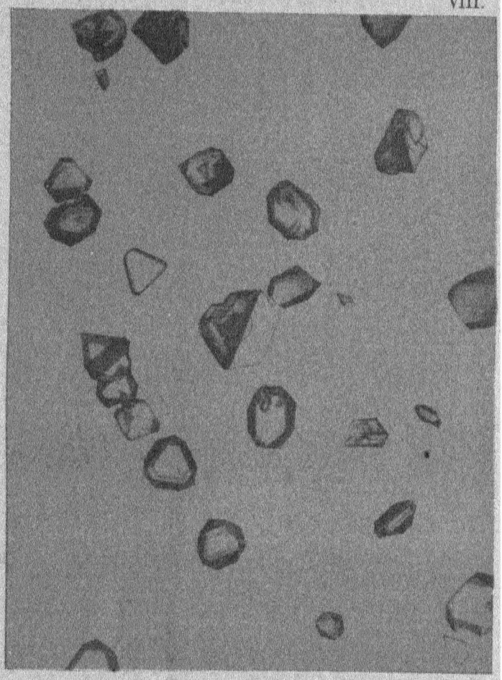
Treatment.—(*a*) Elimination. This should be chiefly relied on. Vomiting should be encouraged, and copious draughts of warm water given. It may be necessary to administer emetics, or use the stomach-pump. (*b*) Prevention of action. Hydrated ferric oxide should be given in considerable quantity. It is estimated that thirty-two parts by weight of this antidote are required to render insoluble one part by weight of arsenious oxide. The antidote must be freshly prepared, as it loses its power if kept for any length of time. To prepare it, a quantity of a solution of a ferric salt, *e.g.*, ferric chloride, should be either rubbed up in a mortar with magnesia, or precipitated by solution of ammonia; in the latter case, the precipitate must be washed on a calico filter before administration. (*c*) Counteraction of effects. This indication must be carried out on general principles. Demulcents should be given to allay irritation, and other symptoms treated as they arise.

Fatal dose.—Minimum for an adult, under circumstances favourable to the action of the poison, two to three grains of arsenious oxide. Cases, however, of recovery, under free vomiting, are recorded from doses of one to two ounces of arsenious oxide. In one very exceptional case of recovery from a large dose (*Case* B 13), the poison swallowed—two masses of arsenious oxide, weighing together 105 grains—was passed per anum. It has been found that persons in the habit of taking small doses of arsenic daily, gradually increasing the quantity, become ultimately able to swallow as much as four to six grains of arsenious oxide, without experiencing the slightest symptoms of poisoning. This habit of "arsenic eating" is practised by the peasants of Styria under the belief that it increases the respiratory powers. Chevers[1] points out that the same habit prevails to a certain extent in the Punjaub, arsenic being there eaten either as an alternative to opium eating, or as an aphrodisiac. It has been noticed, in the case of horses, that the administration of arsenic, in small doses daily, improves their condition, probably by stimulating digestion

Arsenical compounds commonly used as poisons in India.—In the great majority of cases arsenious oxide is employed; in a few the sulphides (orpiment and realgar) are used, either alone, or mixed with arsenious oxide. In exceptional cases, the arsenites of copper are employed (see *Table XV.* 3; *Appendix*, p. 503).

[1] Medical Jurisprudence, p. 117.

VIII.



Page 147. SUBLIMATE OF ARSENIOUS OXIDE CRYSTALS × 63.

From Micro-Photograph by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

Arsenious Oxide (As_2O_3).

Common white arsenic, **Phatkya somul, Sankhya,** or **Somul khar.** This is yearly imported in large quantity, chiefly from the Persian Gulf, and is readily purchaseable all over India.[1] The chief legitimate uses to which it is put in India appear to be as follows:—(1) As a preservative agent, especially for wood. Chevers mentions that with this object it is thrown into the holds of vessels, and placed round wooden foundation piles, and applied to the woodwork and walls of houses. (2) According to Chevers, in preparing the thicker kinds of leather. It seems to be used also to a certain extent for the purposes of preserving skins generally. (3) By goldsmiths in gold-working. (4) For the purpose of destroying rats and other vermin. (5) Medicinally, internally as a cure for fevers, syphilis, and other diseases; and externally as a parasiticide and depilatory. It has already been mentioned that it is used to a certain extent in the Punjaub, as an alternative to opium eating, and as an aphrodisiac.

Homicidal use.—Of all poisons arsenious oxide is the one by far the most frequently employed in India for homicidal purposes. When so employed, the vehicle of administration is most commonly sweetmeat or bread. Sweetmeat, poisoned with arsenic, frequently consists of but little more than sugar or "*goor*," and coarsely-pounded arsenious oxide. Often a club or stick and a piece of flat board, or a couple of stones, are used for pounding the arsenic, and particles of the poison are found adhering to them. When bread is the vehicle used, the coarsely-pounded arsenious oxide is often simply placed between two layers of or mixed with the dough. Sometimes, however, it is ground up with the flour, and the grindstones are found to have particles of the poison adhering to them. In some cases it is the person grinding the flour who adds the poison to it; in others, the poison is put into the handmill by another, during the temporary absence of the person using it. Sometimes the vehicle is cooked vegetable food, *e.g.* cooked rice, pulse, &c. I have also, in cases of alleged poisoning, found arsenious oxide in sweet oil, in tamarinds, in *chuna* (lime) used with betel nut for chewing, and in one case I found it mixed with realgar in a "*beeree*," or native cigarette. Contrary to what one would expect, very often the quantity of the poison contained in an article of food in a homicidal case, is very great (see *Cases* B 14), and the particles of arsenious oxide large enough to be clearly visible. I have even found particles of the poison weighing several grains in a poisoned article of food (see *Case* B 14 *d*). Arsenious oxide, when used for homicidal purposes, is generally

[1] The Bombay Sale of Poisons Act (see p. 112) appears to have the effect of restraining its sale to a certain extent, at any rate in the town of Bombay.

used alone. I have, however, found it in food; with the sulphides of arsenic, with sulphate of copper, with mercuric sulphide, with sulphate of iron, and with pounded glass; and in the Baroda case (*Case* B 21) it was found mixed with diamond dust. Frequently in one and the same case, several persons are poisoned; this was so in 98 out of the 507 cases of arsenical poisoning reported to the Bombay Chemical Analyser, during the ten years ending 1884.[1] Some of these 98 cases of multiple poisoning appear to have been, however, the result of accident. In multiple homicidal cases, very frequently some of the victims are children. In one exceptional year (1878–79), out of thirty-eight persons reported to the Bombay Analyser's Office, as having died from arsenic poisoning during the year, twelve were children. In India, the motives most commonly leading to homicidal poisoning by arsenious oxide, appear to be revenge and sexual passion. Common types of the crime are (1) A. being in enmity with B., gives him (see *Case* B 14 *a*) or his children (see *Cases* B 14 *c* and 15 *b*) some poisoned sweetmeat, or introduces arsenious oxide into his food; of which often others as well as B. partake. As a result a number of persons, often the members of one family, children as well as adults, are poisoned (see *Case* B 15 *a*, &c.); or (2) a wife being anxious to get rid of her husband, puts arsenious oxide—often supplied by a paramour—into her husband's food. Possibly in some cases where husbands are poisoned by their wives, the motive is not homicidal, but connected with the belief in the aphrodisiac virtues of arsenic before referred to. Thus in a case where a woman confessed to having put a white powder (afterwards found to be arsenious oxide) into her husband's food, she alleged that the powder was given to her as a charm, or medicine, "to increase her husband's love for her." In case B 16 *a*, this belief also may have led to the administration of the poison. Case B 16 *b* is a curious example of a poison prepared for a particular individual, going astray and poisoning others. It is seldom that in India acquisition of money or property forms the motive for arsenical poisoning. When theft is the object, dhatura (which see) is the poison usually employed. One or two cases of road robbery, in which arsenious oxide was the poison used, have, however, been reported. In exceptional cases also, superstition leads more or less directly to arsenical poisoning (see *Case* B 16 *c*). It should be borne in mind that the ease with which arsenious oxide can be obtained in India, and the difficulty of tracing its purchase, tends not only to increase the frequency of its use for

[1] Harvey, 'Bengal Medico-legal Report' for 1870–72, states that of 203 cases of poisoning by arsenious acid occurring in Bengal, N.W. Provinces, Oudh, and the Punjaub during the three years, fourteen were cases of multiple poisoning; and of ninety-seven cases of arsenical poisoning reported to the Chemical Analyser, Madras, in the two years 1882–83, twenty-six were multiple cases.

homicidal purposes, but also to facilitate the fabrication of false charges of attempted poisoning. Again also, it must be recollected that an individual, while in custody, may from fear make a false confession of poisoning. In case B 17, there is little doubt but that such a false confession was made.

Abortifacient use.—Arsenious oxide is sometimes given or taken with the object of causing abortion, usually with fatal results. A few of the cases of death from poisoning by arsenious oxide, which have come under my notice in Bombay, appear to have been cases of this kind. Harvey[1] mentions similar cases. In one case, which came before me in 1883, post-mortem examination of a female four months advanced in pregnancy, disclosed a mass of paste containing arsenious oxide, lying in the upper part of the vagina near the os uteri.

Suicidal use.—Suicides by poison in India usually select opium; a certain number, however, use arsenious oxide. From Harvey's[1] statistics, it would appear that in Bengal (including N.W. Provinces, Punjaub, and Oudh) about one-third of the fatal cases of arsenical poisoning are suicidal, but that for one suicide by arsenic there are rather more than seventeen suicides by opium. In Bombay also, about one-third of the fatal cases of arsenical poisoning reported to the Chemical Analyser's Office, appear to be suicidal; and these suicidal cases about equal one-tenth of the total number of suicides by poison, shown in the mortality returns for the whole Presidency. Sometimes, in suicidal cases, the quantity of arsenious oxide found after death in the contents of the stomach, is very large. Taylor[2] refers to a case where the quantity found was four ounces. In one case of suicide, I found 360 grains; and I have several times found over 100 grains. The discovery in the contents of the stomach after death, of a large quantity of arsenious oxide, to a certain extent indicates suicide. It, however, especially in India, where very large quantities are given, or attempted to be given, in homicidal cases, by no means negatives homicide. In case B 14 *c*, for example, the quantity found was 100 grains. Case B 18 *a* is still more conclusive on this point, the victim being an adult instead of a child. Taylor also mentions two other cases, both charges of homicide, in which the quantity found was large (see *Cases* B 18 *b* and *c*).

Accidental cases.—Accidental poisoning from internal administration of arsenious oxide is sometimes met with, generally from the poison being mistaken for some inert mineral substance (see *Cases* B 19). It is also, I think, probable, that in India poisoning by arsenious oxide sometimes arises from the poison having, through carelessness, become by accident mixed with articles of food. The possibility also that in some cases arsenious oxide is intentionally administered without actual homicidal intent, has

[1] Beng. Medico-legal Report for 1870–72. [2] Med. Jurisp., ii., p. 270.

already been alluded to (see *Case* B 16 *a*). Accidental poisoning from external application of arsenious oxide, also occasionally occurs. In the case referred to at p. 145, in which death did not take place for two years, the poison was applied in solution to the skin for the cure of itch, its application being followed by an erysipelatous eruption. Cases B 20 *a* and *b* are additional examples of this form of accidental poisoning. It may be here pointed out that the continued application of arsenious oxide to the unbroken skin, may cause an erysipelatous or eczematous (see *Case* B 20 *c*) eruption thereon. This may be followed by denudation of the epidermis, and sloughing and ulceration owing to the caustic action of the poison. The question as to what effect long-continued application of arsenious oxide has on the unbroken skin, arose in the Baroda case (*Case* B 21).

Cattle poisoning by arsenious oxide.—A large number of horned cattle are yearly poisoned in India.[1] With very few exceptions the poison used is arsenious oxide. Thus, as may be seen from Table XV. 6 (*Appendix*, p. 504), arsenious oxide was found in 714 out of 743 cases of cattle poisoning, reported to the Bombay Chemical Analyser's Office during the ten years ending 1885. Nearly always, where the animals killed are horned cattle, they are poisoned for the sake of their skins.[2] The usual plan adopted is to make some powdered arsenious oxide into a paste with oil, and insert a mass of this, often wrapped in paper, into a cavity scooped out of a head of jowari or other grain, or enclose it in a bundle of grass. In a few cases, the arsenious oxide is mixed with one or other of the following:—orpiment, realgar, red lead, litharge, or pounded glass. Cases of horse poisoning also sometimes come under notice. The motive for these appears generally to be to gratify a spite against the owner of the animals. In one case of horse poisoning, occurring in Bombay, a native gentleman lost seventeen horses in three months. The bodies of the first fifteen were not examined, but the sixteenth and seventeenth were both found to have been poisoned by arsenious oxide. For the symptoms caused in cattle by administration of arsenious oxide and other poisons (see *XVI.*, *Appendix*, p. 504).

Form.—Arsenious oxide is met with in the form of—(*a*) White masses looking somewhat like lumps of white earthenware; these are at first translucent, but become opaque by keeping. (*b*) A more or less fine powder composed of irregular shaped fragments, obtained by mechanically powdering form (*a*): and (*c*) A fine

[1] In 1884, 292 head of cattle were reported to the Indian Chemical Examiners as killed by poison. In Bombay, 677 animals, almost all horned cattle, were killed by poison during the ten years ending 1884.

[2] By *Chamars* or *Chucklers*, i.e., leather-workers; or by *Mahars* or *Dhers*, who claim the bodies of animals dying of disease. Gribble remarks that he has found ordering the bodies to be buried in quicklime effectually stops cattle-poisoning in districts where this crime prevails (Med. Jurisp., p. 212).

powder composed entirely or almost entirely of minute crystals. Forms (*a*) and (*b*) are the forms in common use in India; form (*c*) is only occasionally met with. The difference in appearance under the microscope of forms (*b*) and (*c*) was a matter of much importance in case B 22. Powdered arsenious oxide is usually met with in England, coloured with soot or indigo, as directed under the Arsenic Act (14 Vic. cap. 13, see p. 111),[1] and in a case of poisoning it may be of importance to note the nature of the colouring matter found mixed with the arsenious oxide.

Properties.—**Taste** either nil, or slightly roughish or sweetish; not strongly metallic, as is the case with many white irritant poisonous powders, or acid like oxalic acid. Specific gravity about 3·7. A pinch of powdered arsenious oxide weighs about 17 grains, a teaspoonful about 150 grains, and a tablespoonful about 530 grains (Taylor). **Solubility.**—Cold water dissolves about half a grain to a grain per ounce. Boiling water dissolves more, and water boiled for an hour with it will take up about 12 grains per ounce.[2] A much larger quantity of powdered arsenious oxide than this, may however be suspended in mucilaginous fluids. When finely powdered arsenious oxide is mixed with water, a small quantity rises to the surface and floats thereon, forming a film. The fact that such a film was observed on a particular fluid supposed to have contained the poison administered, may be an important piece of evidence. Alkalies and alkaline carbonates, owing to the formation of alkaline arsenites, augment, and, according to Taylor, organic matter as a rule decreases, the solubility of the poison. Arsenious oxide is very soluble in hydrochloric acid. **Effect of heat.**—Solid arsenious oxide when heated volatilises without charring or fusion. Its vapour received on a moderately heated surface, condenses in minute crystals of characteristic appearance, the majority of which are more or less perfect octahedra (see Fig. 8).

Detection.—(1) By the effect of heat on the solid as stated above. (2) Boiled with water the solution of arsenious acid so obtained: (*a*) acidulated with hydrochloric acid yields a yellow precipitate with hydrogen sulphide, soluble in solution of ammonia; (*b*) yields a yellow precipitate with ammonio-nitrate of silver;[3] (*c*) gives a green precipitate with ammonio-sulphate of copper;[4] and (*d*) the solution (or the powdered solid), boiled with nitric acid and the fluid evaporated to dryness, yields a residue of arsenic acid,

[1] Although the Sale of Poisons Act (see p. 112) has been in force in Bombay for nearly twenty years, I can only recollect one case in which on examination I found powdered arsenious oxide mixed with one of the colouring materials mentioned in sect. 17 of the Bombay Act.

[2] The absence of severe symptoms in case B 13 is no doubt attributable to the sparing solubility of the poison.

[3] Prepared by adding ammonia to silver nitrate solution, in quantity just sufficient to dissolve the precipitate at first thrown down.

[4] Prepared from copper sulphate solution, in a similar way.

which when dissolved in water gives a red brown precipitate with solution of silver nitrate. (3) Powdered arsenious oxide mixed with powdered charcoal and powdered sodium carbonate,[1] and heated in a narrow tube (the "*reduction process*"), yields a sublimate of metallic arsenic as a ring, hair-brown in colour where the film is thin, and dark grey or black and lustrous where thicker. Such a ring cut off and heated in a wide tube, sublimes readily with formation of arsenious oxide, which condenses on the side of the wide tube, in minute crystals of the characteristic appearance before noted. The wide tube should first be warmed above the metallic ring, as arsenious oxide is apt to condense on a cold surface as an amorphous powder. This "reduction process" may be used for the identification of other solid dry arsenical compounds.

Sulphides of Arsenic.

Two of these are in common use in India, viz.: the yellow sulphide, **Orpiment, King's yellow** or **Hurtal, As_2S_3**, and the red sulphide, **Realgar, Sandaracha** or **Munseel, As_2S_2**. Both appear to be favourite medicines of the *hakims*, and to be in common use as depilatories. The yellow sulphide is also largely used in India as a pigment. Both sulphides as met with in commerce, usually contain much arsenious oxide, and are more or less actively poisonous according to the quantity of arsenious oxide present. Taylor seems to attribute the poisonous properties of the sulphides, entirely or almost entirely to the presence of this impurity. In India the sulphides of arsenic are used for criminal purposes much less frequently than arsenious oxide. Harvey mentions that in Bengal, &c., during the three years ending 1872,[2] 223 cases of human poisoning by arsenious oxide were reported, as against seventeen by the sulphides (all by orpiment). In Bombay, during the ten years ending 1884, sulphides of arsenic were detected in less than 6 per cent. of the total number of cases of arsenical poisoning brought to the notice of the Chemical Analyser to Government.[3]

Human poisoning by the Sulphides.—None of the seventeen cases mentioned by Harvey (see above) were homicidal; nearly all appear to have been cases of suicide. Chevers,[4] however, gives details of two cases of attempted homicide by orpiment introduced into food; and Waddell[5] mentions three fatal cases of poisoning by orpiment, of which two at least appear to have been homicidal, as having been brought to his notice in Bengal in one year (1884). I have (in Bombay) met with a few cases of poisoning or attempted poisoning by the sulphides. Some of

[1] Black flux. [2] Report before cited, pp. 245, 260.
[3] See Tables XV. 3 and 6, Appendix, p. 503.
[4] Med. Jurisprudence, p. 123.
[5] Bengal Chemical Examiner's Report for 1884.

these (see *Cases* B 23) were cases of homicide or attempted homicide; the poison, in a few cases realgar, in a few others orpiment, and in a few the mixed sulphides, having been given, or attempted to be given, in articles of food.

Abortifacient use.—Orpiment seems to be sometimes employed as an abortifacient, or ingredient of abortifacient preparations. In one of the seventeen cases reported by Harvey, orpiment appears to have been taken with the object of procuring abortion. In several cases which have come before me in Bombay, I have found orpiment in packets discovered in the possession of persons charged with procuring abortion; and in one or two cases in powders stated to have been given with this object. In one of these last-mentioned cases, the poison was apparently present in the form of sulpho-arsenite of calcium. In one or two cases also I have found mixed arsenious oxide and orpiment, in the paste used for arming abortion sticks (see p. 376). In another case in which it was stated that abortion had been procured by the local application of drugs, a board used for mixing, and two stones used for grinding the drugs employed, were found to be stained with realgar and red lead.

Realgar as an adulterant in opium.—In a few cases I have found realgar in small quantity, in opium seized in the possession of vendors of the drug, charged with selling adulterated opium.[1] I am not aware, however, that any cases of arsenical poisoning have arisen from this adulteration.

Use as cattle poisons.—The sulphides are occasionally, but not often, used as cattle poisons. During the last ten years in Bombay, of the cases of cattle poisoning in which arsenic was detected, in about 1½ per cent. sulphides only, and in a somewhat smaller number mixed arsenious oxide and sulphides, were found. Of the two sulphides, orpiment appears to be more commonly used than realgar.

Detection.—When pure, the sulphides are practically insoluble in water and hydrochloric acid. Heated *per se*, they yield a mixed sublimate of sulphide and oxide. Their identification is best effected by the reduction process, converting the ring of metallic arsenic obtained into arsenious oxide, to which, after solution in boiling water, the liquid tests for arsenious oxide may be applied.

Arsenites of Copper.

Two of these are in common use as pigments, viz. Scheele's green $Cu\,H\,As\,O_3$ (acid cupric arsenite) and Schweinfurth, or emerald green (aceto-arsenite of copper), $3\,Cu\,As_2\,O_4 + Cu\,(C_2\,H_3\,O_2)_2$. Both are known in India under the name "*Hirwa.*" Both, although insoluble in water, are readily dissolved by the acid

[1] Bombay Analyser's Report, 1886.

fluids of the stomach, and when absorbed give rise to the usual symptoms of arsenical poisoning. They are seldom used in India either by homicides or suicides. I have, however (in Bombay), met with two instances of alleged attempt at homicide by arsenite of copper (see *Cases* B 24 *a* and *b*); and a case of suicide by arsenite of copper was reported a short time ago in Bombay city. Cases of poisoning by the arsenites of copper, usually arise either from their being used (in ignorance of their poisonous nature) to colour articles of food or confectionery; or from their accidental absorption into the system, from other articles coloured by them. As regards the first class of cases, I once met with a case where a whole family were poisoned by "*Halwa*" thus coloured, and Taylor records a fatal case arising from eating blancmange, coloured with arsenite of copper. Again, in several instances, I have found arsenite of copper used to colour the small sugar-plums known as "*til-gul*," which are sold at the *Makar Sakrant* festival[1]; and Dr. W. Center states that the confectioners of Lahore use arsenite of copper to colour their wares.[2] Taylor mentions several instances of children being poisoned by confectionery coloured with these compounds. Cases of the second class are often cases of chronic or slow poisoning, arising from inhalation of particles of the poison, detached from wall-papers coloured with arsenical green. Chevers mentions one such case as having occurred in India, also another where the poisoning resulted from sleeping in a bedroom, the walls of which had been coloured with arsenite of copper, loosely put on with size.[3] Children again are sometimes poisoned, by sucking green arsenical paint off toys (see *Case* B 24 *c*).

Detection.—Both arsenites of copper are soluble in solution of ammonia, with formation of a blue liquid, which on evaporation re-deposits the compound. Both also are soluble in hydrochloric acid, the solution yielding (1) evidence of the presence of copper to the usual liquid tests for this metal (see copper), and (2) evidence of the presence of arsenic to Reinsch's process (which see p. 158). The presence of arsenic in these compounds may also be demonstrated by the reduction process as directed for the sulphides. The presence of acetic acid in the aceto-arsenite may be recognised by heating the compound with sulphuric acid, when free acetic acid is liberated; or with sulphuric acid and alcohol, when acetic ether is evolved.

Other Poisonous Arsenical Compounds.

Fly powder.—This is a black powder, consisting of, or containing, metallic arsenic, partly converted into arsenious oxide.

[1] January 12th or 13th. In one instance I found in "*til-gul*," from a vendor's stock; green, red, and yellow sugar-plums, coloured respectively with aceto-arsenite of copper, mercuric sulphide, and chromate of lead.

[2] Indian Medical Gazette, 1874, p. 116. [3] Med. Jurisp., p. 125.

A few cases of poisoning by it have been recorded in France and Germany.

Arseniuretted Hydrogen, AsH_3.—This highly poisonous gas is evolved when hydrogen is set free from materials, or in liquids, containing arsenic (see *Marsh's process*). A few cases of poisoning by its inhalation, all accidental, have been reported.

Alkaline arsenites.—These are more soluble in water than arsenious oxide, hence the greater solubility of the latter in alkaline liquids. Cases of poisoning have been recorded from Fowler's solution (Liquor Arsenicalis B.P. and I.P.) This is a weak solution of potassium arsenite, coloured with compound tincture of lavender; strength, I.P., four grains of arsenious oxide to the ounce;[1] or B.P. of 1885, 4·375 grains to the ounce, or 1 per cent. Cases (see B 20 *c*) have also occurred from the use of arsenious oxide, mixed with potassium carbonate solution (potassium arsenite), as a sheep wash; and Taylor mentions a case in which 340 school children were poisoned, by drinking tea made with water from a boiler into which mixed arsenite and arsenate of sodium had been put, in order to cleanse it from deposit.

Alkaline arsenates.—Salts of the acids derived from arsenic anhydride As_2O_5. Potassium and sodium arsenates. These rarely give rise to cases of poisoning. Taylor[2] refers to three accidental cases, and gives details of one attempt at homicide; in the latter, arsenate of potassium was given in wine. Paper soaked in solution of an alkaline arsenate mixed with sugar, is used for poisoning flies, under the name of "Papier Moure." Arsenate of potassium is used for preserving skins, and has lately been imported into India for this purpose. Chevers[3] mentions two cases (both in the Punjaub) of attempted cattle poisoning, by pieces of grass moistened with solution of potassium arsenate.

Arsenic in dyeing materials.—A large number of dyes, of various colours, are obtained from rosaniline, a red colouring matter prepared by the action of an oxidising agent (usually arsenic acid), on aniline. Red aniline dyes have been found to contain arsenic, and workmen engaged in their manufacture have been known to suffer from cutaneous eruptions, and the usual symptoms of chronic arsenical poisoning. Cases also are reported of cutaneous eruptions arising on the feet, apparently due to wearing socks dyed red with aniline colours (see also p. 238). Taylor further mentions that arsenate of aluminia is used by dyers as a mordant with various colours, and that fabrics dyed in this way have been found to contain arsenic.

Liquid reactions of arsenic acid.—Arsenic acid differs from arsenious acid in giving, (1) a red-brown precipitate with silver nitrate, and (2), in moderately strong solution, a precipitate with a mixture of ammonia, ammonium chloride, and magnesium

[1] This was the strength of the preparation of the B.P. of 1867.
[2] On Poisons, p. 351. [3] Med. Jurisp., p. 133.

sulphate solutions. An acidulated solution of arsenic acid is precipitated by sulphuretted hydrogen much more slowly than a similar solution of arsenious acid.

Detection of Arsenic in Viscera, &c.

In the presence of organic matter, the ordinary tests for the recognition of arsenic become inapplicable. Further, the ready volatility of arsenic precludes incineration being employed for the destruction of organic matters mixed with it. Hence to separate arsenic from organic matter, and bring it into a form in which it may be readily recognised, special processes are required. Before describing these, certain points bearing on the inferences to be drawn from the analytical results must be considered.

1. **Disappearance of arsenic from the body (*a'*) after death.**—Arsenic, like other inorganic poisons, cannot disappear by putrefaction. Hence it may be detected in human remains after any period of interment. Its antiseptic power, and the sparing solubility of its most commonly-used compounds, tend specially to favour this. On the other hand, arsenic, being volatile at the temperature of combustion, disappears when a body is burnt. It may, however, be, and has been, detected in partly-burnt bodies. (*b'*) **Disappearance during life.**—During life, vomiting and purging tend to free, first the stomach, and subsequently the intestines, from the poison. In exceptional cases, complete disappearance from the contents of the stomach may take place very rapidly. Taylor[1] mentions a case where this occurred within twenty-four hours; and I once met with a case in which arsenic could not be detected in the contents of the stomach of a woman, who died from arsenical poisoning in six hours.[2] On the other hand, two cases are recorded, one of death in six, and the other of death in seven days, in both of which arsenic was found after death in the contents of the intestines.[3] During life also, absorption of the poison takes place very rapidly. Taylor found it in comparatively large quantity in the liver in a case of death in three hours.[4] Again, during life, absorbed arsenic tends to undergo elimination from the body, by the kidneys and other emunctories. Complete elimination may undoubtedly take place in fifteen days, and may, there is good reason to believe, take place even in a shorter time than this (see *Cases* B 25 *a* and *b*). On the whole, therefore (1), Entire absence of arsenic from the body is quite consistent with the supposition of death (after some days) from arsenical poisoning; it, however, strongly indicates survival for some days; and

[1] On Poisons, p. 335.

[2] Bombay Analyser's Report, 1879-80; the poison was given in food; arsenic was detected in the liver.

[3] Taylor on Poisons, p. 336. [4] *Ibid.*, p. 334.

(2) In a case of death from arsenical poisoning, absence of arsenic from the alimentary canal is a moderately strong indication that the individual lived some time after swallowing the poison, the indication being stronger if arsenic is found to be absent from the contents of the intestines, as well as from the contents of the stomach.

2. **Conversion in the body of arsenious oxide into yellow sulphide of arsenic.**—It has already been pointed out that this may occur; the reverse change cannot, however, take place. The discovery, therefore, of arsenious oxide in the body, shows that the poison administered contained arsenious oxide. On the other hand, the discovery of yellow sulphide of arsenic in the body does not prove that the poison was administered in the form of yellow sulphide.

3. **Presence of arsenic in earth.**—Arsenic is sometimes found in minute quantity in earth, but has hitherto only been found in earth in a form insoluble in water. It has been alleged, however, that arsenical earth may, under the action of the air, yield a soluble arsenical compound. Further, it has been found, that when arsenic in solution is introduced into the alimentary canal of a dead body, post-mortem imbibition takes place, and arsenic, passing through the walls of the alimentary canal, becomes imbibed by tissues external to, but in contact therewith. In the case, therefore, of an **exhumed body**, in the viscera of which arsenic has been detected, it may be alleged that the arsenic found therein was derived from the surrounding earth. Either of two cases may arise. The parietes of the body may be found (1) **intact**, or (2) **not intact**, and the viscera more or less mixed with earth. In case (1), the discovery of any notable quantity of arsenic in the body completely negatives the theory of earth derivation. The presence of traces even can hardly be accounted for in this way. In case (2) the earth derivation theory is in the highest degree improbable, if a notable quantity of **soluble arsenic** is detected. Whenever, however, case (2) arises, a portion of the surrounding earth should always be submitted to analysis, and even in case (1) it is advisable to preserve a portion of the surrounding earth, so that it may be examined for arsenic, should traces only thereof be found in the body. In India **vomited matters** are frequently found mixed with earth; here, again, it is important to ascertain whether or no arsenic present in such matters, is present in a form soluble in water. Should soluble arsenic be present, and especially if it be present in notable quantity, earth derivation is improbable. Earth and gravel are often found in the stomachs of **cattle**. Hence the presence of a minute quantity of insoluble arsenic in the bodies of such animals, may possibly be accounted for on the theory of earth derivation.

Quantity of arsenic found.—The quantity of arsenic found in the viscera of an individual may, to a certain extent, affect

(*a'*) the presumption as to suicide or homicide; or (*b'*), the presumption as to the cause of death. Question (*a'*) has already been considered (see *ante*, p. 149). Question (*b'*) also has been discussed with reference to poisons generally (see *ante*, p. 126). Points bearing specially on the possibility of a minute quantity of arsenic being discovered in the viscera, in a case of death from causes other than arsenical poisoning, are—(1) the existence of the habit of arsenic eating, and the frequent use by *hakims* of arsenic in the treatment of disease (see *ante*, p. 147); (2) the possibility of earth derivation just discussed; and (3) the fact that arsenic is *not* a natural constituent of the body, although the contrary was at one time asserted. It must not be lost sight of, also, that arsenic may be present in small quantity as an impurity, in drugs administered for medicinal or other purposes. Antimony and bismuth[1] compounds are liable to contain traces of arsenic; so, also, is sulphuric acid, and it has already been mentioned that realgar has been found in opium. As regards the question of arsenic as an impurity in reagents, see below.

Processes for the Separation of Arsenic from Organic Mixtures.

The principal processes employed for the separation of arsenic from organic mixtures, are: (1) Deposition as metallic arsenic on copper, or Reinsch's process; (2) Separation as arseniuretted hydrogen, or Marsh's process; (3) Separation by distillation as chloride of arsenic; and (4) Separation by precipitation as sulphide of arsenic.

Many of the reagents used in the above process, *e.g.*, sulphuric acid, hydrochloric acid, metallic zinc and metallic copper, are specially liable to contain arsenic. All should, therefore, be ascertained to be arsenic-free before use. As regards metallic copper, however, see Reinsch's process.

Reinsch's process.—This consists in boiling the suspected liquid acidulated with about one-fourth of its volume of hydrochloric acid (or solid matters cut into small pieces and mixed with hydrochloric acid diluted with about four volumes of water), with a succession of pieces of clean, bright, metallic copper foil or gauze. If arsenic is present, a steel-grey or black stain—an alloy of arsenic and copper—forms on the surface of the foil or gauze. The stained pieces of copper are then washed, dried, and

[1] Dr. Richardson, in the case of *R.* v. *Smethurst* (case B 26 *b*), stated that in the bismuth usually administered in medicine (sub-nitrate), he had found nearly half a grain of arsenic per ounce, and that in one case, where for dyspepsia five grains of bismuth had been given three times a day for six days, he had found about a fiftieth of a grain of arsenic in the patient's urine. Dr. Thudichum, at the same trial, stated that he had found both arsenic and antimony in subnitrate of bismuth, and also in grey powder.

heated in a test-tube,[1] when the stain, if arsenical, disappears, and a sublimate of arsenious oxide is obtained, which, under the microscope, is found to consist of minute crystals of the characteristic appearance before noted. Unless such crystals are obtained, the presence of arsenic has not been demonstrated, because the staining of the copper may be due to (1) the action of organic matter only, or the formation of sulphide of copper; or (2) the deposition on the copper of metals other than arsenic, *e.g.*, mercury, antimony, silver, bismuth, &c.

Under the circumstances of the process however, of the metals which deposit on copper, two only yield sublimates, viz., mercury, which yields a sublimate of minute globules of metallic mercury, and antimony, which yields a non-crystalline sublimate.

Reinsch's process is the most generally applicable of all. It cannot, however, be used in cases where the liquid contains matters which dissolve the copper, *e.g.*, nitrates, chlorates, or ferric chloride. It is only also in such cases that the presence of arsenic in metallic copper can lead to error.[2] Hence, if the copper dissolves, or the deposit forms only very slowly thereon, one of the other processes should be resorted to.

Marsh's process.—This consists in introducing a liquid suspected to contain arsenic, into a vessel from which hydrogen gas is being evolved. If arsenic be present, the nascent hydrogen attacks it, forming arseniuretted hydrogen. The arsenical nature of the gas evolved may be proved: (1) By passing it through a narrow hard glass tube, heated to redness for a portion of its length; when a deposit of metallic arsenic forms in the cool part of the tube beyond the heated portion. (2) By igniting the gas, and holding a piece of cold white porcelain in the flame, when a deposit of metallic arsenic forms thereon; (3) By passing the gas through solution of silver nitrate, when a black deposit of metallic silver is thrown down, and the arsenic is converted into arsenic acid, which remains in solution.

The arsenical nature of these products of the process is proved as follows:—(1) The portion of the narrow tube containing the deposit or ring of metallic arsenic, is heated, in a wide tube, like the metallic ring in the reduction process. It volatilizes readily, yielding a similar crystalline sublimate. (2) The spots on porcelain, where thin, are seen to be hair-brown in colour, and (*a*) are soluble in chloride of lime solution, (*b*) are insoluble in stannous chloride solution, and (*c*) dissolve in aqua regia; the solution, when evaporated to dryness, yielding a residue of arsenic acid, which gives a brick-red precipitate with silver nitrate solution.

[1] In the same way as the metallic ring in the reduction process.

[2] Unless such substances are present, the copper does not dissolve during the process. If the copper remains undissolved and becomes rapidly coated, an arsenical deposit formed on the copper cannot be due to the presence of arsenic in the copper employed.

(3) The silver nitrate solution is treated with excess of hydrochloric acid, filtered, and the filtrate evaporated to dryness; silver nitrate solution added to the residue, gives a brick-red precipitate.

The apparatus used may be an ordinary gas-bottle, such as is employed for preparing hydrogen gas, fitted with a tube filled with fused calcium chloride (for the purpose of drying the gas evolved), to which is attached a long narrow hard glass tube, ending either in a jet, or a downward bend. The materials used may be zinc and dilute sulphuric acid, or zinc and hydrochloric acid. A quantity of hydrogen gas must be allowed to escape before heat is applied to the narrow tube (avoidance of explosion). Before the suspected liquid is introduced, the narrow tube must be heated to redness for about fifteen minutes. If no deposit forms, the materials are arsenic-free. In this way the purity of the hydrochloric acid used in Reinsch's, and other processes, may be ascertained. **Bloxam's** modification of Marsh's process consists in evolving the hydrogen gas required, by electrolytic decomposition of water acidulated with sulphuric acid. In this way the use of zinc (which frequently contains arsenic) is avoided. In another modification, **Fleitmann's**, applicable to arsenic but not to antimony, the hydrogen is evolved by heating zinc with potassic hydrate solution.

Marsh's process is not applicable to the sulphides of arsenic, or to solutions containing arsenic as arsenic acid, or as an arsenate (see however below).

The choride distillation process.—The substance under examination (cut into small pieces if a solid) is first thoroughly dried on a water-bath. The dry residue is then distilled to dryness with the strongest obtainable hydrochloric acid, and the residue in the retort distilled again to dryness with a little more of the same acid. Under these conditions, arsenic, if present, distils over as chloride of arsenic, and may be recognised in the distillate, by subjecting this to Reinsch's or Marsh's process.

Precipitation as sulphide of arsenic.—Mixtures containing little organic matter may be boiled with dilute hydrochloric acid, filtered, and the filtrate precipitated by washed sulphuretted hydrogen. If much organic matter be present, this must be first destroyed by boiling the substance under examination (cut into small pieces if a solid) with dilute hydrochloric acid, and adding to the boiling liquid from time to time, a small quantity of crystallized potassic chlorate. The organic matter having been destroyed, the liquid is filtered, sulphurous acid added to it,[1] and washed sulphuretted hydrogen passed through it. The precipitated sulphide is separated by filtration, washed, dissolved in ammonia, the solution filtered, and the filtrate evaporated to

[1] To reduce the arsenic acid. Marsh's process may be applied to the detection of arsenic acid and the arsenates, if these be first treated with sulphurous acid.

dryness. The residue of impure sulphide of arsenic may then be subjected at once to the reduction process, or (for quantitative determination of the arsenic present) treated as follows:—Boil with strong nitric acid, evaporate to dryness, dissolve in a little water, filter, and add to the filtrate a mixture of solutions of magnesium sulphate, ammonium chloride, and ammonia. After twenty-four hours, filter, wash the precipitate with ammonia-water, dry and weigh. It consists of ammonium magnesium arsenate, and contains 39·47 per cent. of metallic arsenic.

CHAPTER XI.

VOLATILE METALS (*continued*). ANTIMONY AND MERCURY.

ANTIMONY, Sb = 122.

ANTIMONIAL poisoning—extremely rare in India—usually arises from the potassio-tartrate (tartar emetic). A few cases also are recorded of poisoning by the chloride (butter of antimony).

Tartar Emetic, $2C_4H_4O_6(SbO)K+H_2O$.

This, also called potassio-tartrate of antimony and tartarized antimony, produces effects on the system very similar to those produced by arsenious oxide. Unlike the latter, however, it has a strong metallic taste. Tartar emetic has a very marked depressant action on the heart, and a depressant action on the nervous system generally. Hence, in poisoning by it **prominent symptoms** are—extreme faintness, collapse, and muscular weakness. Loss of voice has been noticed, and convulsions often precede death. In exceptional cases (as in arsenical poisoning), vomiting is slight or absent, necessitating the administration of emetics. Tartar emetic applied to the skin produces a pustular eruption thereon, and may become absorbed, giving rise to constitutional symptoms. A pustular eruption on the skin has also been noticed, in cases of poisoning by internal administration of the drug.

Acute poisoning by tartar emetic is seldom homicidal; it is frequently accidental from the poison being mistaken for some harmless powder, *e.g.*, Epsom salts, or carbonate of soda. It may also be remarked that, although tartar emetic is popularly well known to be a powerful emetic, it is not equally well known to be a powerful poison. It is possible that this may explain the mysterious Bravo case (*Case* B 26 *a*).

Chronic poisoning.—Some remarkable trials for murder, by the administration of repeated small doses of tartar emetic, have taken place in England (see *Reg.* v. *Smethurst, Case* B 26 *b*, and Dr. Pritchard's trial, *Case* B 26 *c*). In some cases of chronic antimonial poisoning, the failure of treatment to control the prominent symptoms, viz., nausea, vomiting, and diarrhœa, with

great depression and muscular weakness, has been the first thing to excite suspicion as to the true nature of the case.

Preparations containing Tartar Emetic.—Vinum antimoniale B.P. and I.P. contains 2 grains of tartar emetic per ounce. Tartar emetic is also contained in small quantity in several quack pills, *e.g.*, Dr. Johnson's, Mitchell's, and Dixon's pills,[1] and forms one-fifth by weight of the Unguentum antimonii tartarati B.P. and I.P.

Fatal period.—Shortest recorded, seven hours (in a female æt. twenty-one).[2] Longest, one year.[3] Usual, ten hours to four days.

Fatal dose.—Smallest (in a child), three-quarters of a grain. Largest non-fatal, half an ounce. Taylor considers that, under circumstances favourable to the action of the poison, ten to twenty grains taken at once, might prove fatal to an adult; but that, if taken in divided doses, a smaller quantity might suffice.[4]

Post-mortem appearances.—Similar generally to those of arsenical poisoning. Aphthous inflammation of the mouth, throat, and gullet, and aphthous ulceration of the small intestines, have been observed.

Treatment.—The usual treatment for irritant-poisoning, with the administration as an antidote of an infusion containing tannin, *e.g.*, solution of tannic acid, decoction of oak-bark or of cinchona bark, or strong green tea. Stimulants may be required to counteract depression.

Other Antimonial Compounds.

Trichloride, or Butter of Antimony, $SbCl_3$.—A strong solution of this is used for browning gun-barrels; also sometimes in surgery as a caustic; and in pharmacy as a source of oxide of antimony. It is highly corrosive, giving rise, when swallowed, to symptoms of corrosive poisoning, plus constitutional effects similar to those caused by tartar emetic. A few fatal cases of poisoning by the chloride of antimony are recorded. In one of these narcotism succeeded the usual irritant symptoms. The post-mortem appearances are those of corrosive poisoning.

Antimony trioxide, Sb_2O_3, although insoluble in water, is soluble in the fluids of the stomach. It is used in medicine in the form of pulvis antimonialis—an imitation of the old James's powder—a mixture of one part of oxide of antimony to two of phosphate of lime. Its action on the system is similar to that of

[1] About one-sixteenth to one-twenty-fifth of a grain in each pill (Blyth on Poisons, p. 547).
[2] Wormley on Poisons, p. 218.
[3] Guy's For. Med. p. 487.
[4] On Poisons, p. 464.

tartar emetic, but milder in degree. Overdoses of it have given rise to dangerous symptoms.

Antimony trisulphide, Sb_2S_3.—This is met with in two forms: (1) as a black crystalline mass or powder—native sulphide of antimony, black antimony, or "surma";[1] and (2) as an orange-coloured powder—precipitated sulphide of antimony. Although pure sulphide of antimony is probably inert, the following points of medico-legal interest attach to it:—(1) Commercial black sulphide of antimony frequently contains arsenic, hence—if not first thoroughly purified—tartar emetic prepared from it is liable to contain arsenic. (2) The orange sulphide of antimony, plus a variable proportion of antimony trioxide, forms the antimonium sulphuratum of the B.P. and I.P., and this forms about one-fifth by weight of Plummer's pills (Pilula hydrargyri subchloridi, composita, B.P. and I.P.).[2]

Detection of Antimony.

Antimony after absorption is eliminated mainly by the urine, and may be detected during life in this fluid. It is probably eliminated from the body at least as rapidly as arsenic. Like arsenic also, antimony does not disappear by decomposition, and has been detected in the body after long periods of interment.

Tartar Emetic.—This is a white crystalline powder which, when heated, chars, yielding, if a spirit-lamp only be used, no metallic or crystalline sublimate. It is almost insoluble in alcohol, ether, and chloroform. One part dissolves in fifteen of water at 58° F., and in two parts of boiling water. The solution (1) on evaporation yields tetrahedral crystals; (2) gives no precipitate with potassium ferrocyanide; (3) gives a dirty yellow white precipitate with infusions containing tannin; (4) if concentrated, gives, with strong hydrochloric acid, a white precipitate soluble in tartaric acid, and in excess of hydrochloric acid; (5) acidulated with hydrochloric acid, and treated with sulphuretted hydrogen, yields an orange-coloured precipitate of antimony sulphide insoluble in ammonia, but dissolving on digestion with sulphide of ammonium.

Sulphide and Oxide of antimony dissolve on boiling with strong hydrochloric acid, the solution (and solution of chloride of antimony) yielding: (1) with water, a white precipitate soluble in tartaric acid; and (2) when moderately diluted and treated with hydrogen sulphide, a precipitate of antimony sulphide, of the characters stated above.

Detection of Antimony in organic mixtures.—To Reinsch's

[1] Used in India as a collyrium. Women, according to Balfour (Cyclopædia, iii., 248), always use kohl or lamp-black instead of surma.

[2] The presence of antimony as an occasional impurity, in grey powder and subnitrate of bismuth, has already been noticed (see note, p. 158).

process, antimony, like arsenic, yields a deposit on copper, which, however, when heated, sublimes only at a high temperature, yielding an amorphous sublimate of Sb_2O_3. For Reinsch's process in the case of antimony, the **Galvanic deposition process** may be substituted. This consists in placing in a concentrated hydrochloric acid solution of the matters under examination, a slip of platinum foil, with a piece of pure zinc in metallic contact therewith. Metallic antimony is deposited on the platinum. The stained platinum is washed, boiled with nitric acid, the acid solution evaporated to dryness, and the residue dissolved in dilute HCl, and tested for antimony by hydrogen sulphide. Deposits of antimony obtained by Marsh's process may be similarly treated. To **Marsh's process** antimony yields deposits distinguishable from arsenical deposits as follows: (1) The metallic ring in the tube is deposited closer to the heated portion, is sublimable only with difficulty, and yields no sublimate of octahedral crystals. (2) The spots on porcelain are smoky black (not brown), insoluble in chloride of lime solution, but slowly soluble in stannous chloride solution. (3) In the vessel containing the silver nitrate solution, the antimony falls (as antimonide of silver) with the deposit, and may be recovered by boiling the deposit for some time with tartaric acid. **Precipitation as Sulphide.**—The organic matter may be destroyed in the same way as when this process is used for separation of arsenic. The precipitated sulphide may then be collected, and dissolved by boiling it with strong hydrochloric acid, and the amount of antimony present estimated volumetrically by a standard solution of iodine; the antimony solution being first treated with sodium tartrate and carbonate, to weak alkaline reaction.[1]

MERCURY, Hg = 200.

Mercurial poisoning may be acute or chronic.—**If acute,** the symptoms may be either those of corrosive, or those of non-corrosive irritant-poisoning. **If chronic,** either salivation or mercurial tremors, or both, may be present; and in two cases of chronic poisoning by an organic mercurial compound (mercuric methide), the brain was specially affected.

Acute mercurial poisoning is rare in India. Accidental cases are, however, occasionally met with, and Chevers mentions one doubtful homicidal case.[2]

[1] Under the conditions stated, Sb_2O_3 oxidizes into Sb_2O_5. The end reaction is the permanent appearance of free iodine, in the liquid under test.

[2] In the case referred to, deceased, a prostitute, died from the effects of an irritant poison administered to her in sweetmeat, by a man afterwards tried and convicted of her murder. It was alleged that the poison used was ruskapoor (see calomel), but the evidence on this point was very unsatisfactory (Chevers, p. 266).

Symptoms.—Acute mercurial poisoning most commonly arises from swallowing corrosive sublimate, in which case, and also when the nitrates are swallowed, the symptoms present are those of corrosive poisoning. In acute cases other than these, the symptoms are those of non-corrosive irritant-poisoning, and vary in severity according to the activity of the compound swallowed. Mercuric compounds are more active than mercurous compounds. Soluble mercurial salts have a strong metallic taste. In corrosive cases, (1) Intense burning pain in the mouth and throat comes on immediately, and (2) The lining membrane of the mouth and throat becomes white and shrivelled. These characters are absent in non-corrosive cases. In both corrosive and non-corrosive cases, the usual symptoms of irritant-poisoning are present, and more or less complete suppression of urine is a common symptom. In some cases coma has been observed. If the case is prolonged, salivation, as in chronic poisoning, may appear; but this is not a common symptom in acute cases.

Post-mortem appearances.—These, according to the case, may be those of corrosive, or those of non-corrosive irritant-poisoning. Perforation of the stomach is rare. The gastric mucous membrane may be found covered with a greyish deposit of metallic mercury, or there may be a black deposit of the sulphide. The intestines and urinary organs are generally much congested. In acute poisoning from external application of mercurial compounds, post-mortem appearances of irritation of the alimentary canal are present.

Treatment.—The usual treatment for corrosive or irritant-poisoning, with the administration as an antidote, of albumen (*e.g.* white of egg), or albuminous fluids.

Chronic mercurial poisoning is liable to arise in persons whose occupation exposes them habitually to the vapours of metallic mercury or its compounds, or to constant contact with mercurial compounds. It may also arise from often repeated small doses of any mercurial preparation, and hence may result from the abuse of such preparations in the treatment of disease. Cases of this last description used formerly to be of frequent occurrence in India.

Symptoms.—These may be debility, nausea, and vomiting, accompanied by colicky pains, and followed by salivation. In other cases, mercurial tremors are the first symptoms to appear.

Salivation.—This commences with a coppery taste in the mouth. The gums swell and become tender and spongy. There is profuse ptyalism, fœtor of the breath, and febrile disturbance. A blue line may be present on the gums. In extreme cases, the tongue and cheeks swell, ulceration appears in the mouth, the jaws become necrosed, and the teeth drop out. Mercury is present in the saliva. **Salivation may—(1) Last for almost any period**: in one exceptional case it is said to have lasted six

years. (2) **Intermit,** and recur after an interval of three months or more. In exceptional cases also, an interval of three months or more has been observed between the discontinuance of mercurial treatment, and the first appearance of salivation. (3) **Appear in acute cases,** but rarely appears in these under twenty-four hours, although one case is reported where it appeared in three hours. (4) **Arise from very small doses.**—Idiosyncrasy in some cases renders an individual specially sensitive to the action of mercury; a case, for example, is recorded where salivation was caused by two grains of calomel. In other cases, idiosyncrasy appears to have the opposite effect. Children, it may be noted, bear mercury better than adults. (5) **Arise from causes other than administration of mercury.**—Thus it has resulted from the administration of compounds of arsenic, antimony, copper, lead, bismuth, and gold; also from administration of iodine, sulphuric acid, hydrocyanic acid, digitalis, cantharides, colchicum, croton oil, opium, carbolic acid, and nitrobenzene, and may occur idiopathically. In non-mercurial salivation, mercury is, of course, absent from the saliva.

Cancrum oris, a disease liable to affect children, especially those that are cachectic or badly fed, has been mistaken for mercurial salivation. Taylor[1] mentions a case in which a child, having died from cancrum oris, a charge of malpraxis was brought against the medical attendant. It was, however, proved that no mercury had been administered.

Mercurial tremors, or shaking palsy.—This as a rule comes on gradually, affecting first the muscles of the arms, and subsequently those of other parts of the body. The affection begins with unsteadiness and quivering, increasing to tremors, which ultimately become so violent as to resemble convulsions. All voluntary movements requiring the aid of the affected muscles, can only be performed by violent starts. In advanced cases, walking, articulation, and mastication, all become affected. Finally there is loss of memory, sleeplessness, delirium, and death. The skin is dry, and has a brown tint. Salivation may or may not be present.

Mercurial Preparations and Compounds.

1. **Corrosive mercurial salts,** viz., corrosive sublimate and the nitrates of mercury.

(*a*) **Corrosive sublimate, Mercuric chloride, $HgCl_2$.—Talachiknasumbul Darchikna** (*Hind.*). This occurs in heavy crystalline masses, or as a white crystalline powder. Heated, it melts and sublimes in prismatic crystals. It is freely soluble in alcohol and ether, and is soluble in sixteen parts of cold or three of boiling water, and more soluble in solutions of alkaline

[1] On Poisons, p. 370.

chlorides than in pure water. The ordinary medicinal dose is one-sixteenth to one-eighth of a grain, and three to five grains may be regarded as a minimum fatal dose. A case of recovery after swallowing an ounce is on record. The usual fatal period is one to five days, but in one case death occurred in half an hour. Corrosive sublimate is readily absorbed through the unbroken skin, and acute poisoning, non-corrosive in character, may arise from its absorption. It is contained, to the extent of half a grain per ounce, in the Liquor hydrargyri perchloridi B.P. and I.P.

(*b'*) **Nitrates of mercury,** viz., normal mercurous $Hg_2 2NO_3$, and normal mercuric $Hg2NO_3$. These salts are freely soluble in water containing nitric acid, but pure water is liable to decompose them, with formation of sparingly soluble basic nitrates. Heated, they decompose, giving off red fumes of nitric oxide, and leave a residue of mercuric oxide. Swallowed, they produce symptoms similar to those caused by swallowing corrosive sublimate. A fatal case from external application of nitrate of mercury in a liniment is recorded; also a case of chronic poisoning from its use as a local application to the neck of the uterus; and another case where a workman whose occupation for four years had been packing skins brushed over with solution of nitrate of mercury, died of chronic mercurial poisoning: others, however, similarly employed in the same factory were unaffected.

2. **Other mercuric compounds.**—(*a'*) **Mercuric cyanide** $Hg(CN)_2$. This is a white soluble salt which, when heated, gives off an inflammable gas (cyanogen), and yields a sublimate of metallic mercury. Although non-corrosive, it is little less active than corrosive sublimate; ten grains has caused death. A case of acute poisoning (non-fatal) from swallowing the **sulphocyanide** $Hg(SCN)_2$, is recorded. This, more correctly mercuric thiocyanate, is sold in little cylinders as a toy under the name of Pharaoh's serpents, so called from the serpentine form of the copious ash yielded by them when burned.

(*b'*) **Subsulphate of mercury, Turpeth mineral** $HgSO_4(HgO)_2$. This is a yellow powder which, although sparingly soluble in water (1 in 2000 in cold, and 1 in 600 of boiling), has a strong metallic taste. Taylor mentions two fatal cases, in which the quantities swallowed were respectively forty and sixty grains.

(*c'*) **White precipitate, Ammoniated mercury. Mercurammonium chloride,** NH_2HgCl.—This is prepared by adding solution of ammonia to solution of corrosive sublimate. It is a white powder which, when boiled with water, becomes yellow. It is soluble in nitric acid, but insoluble in water, alcohol and ether. According to Taylor, it frequently contains mercuric chloride as an impurity. It is only used medicinally as an external application for the cure of parasitic affections. Swallowed, it has caused severe symptoms in several cases, and at

least one death. Chronic poisoning has arisen from its external application. Cases of recovery after swallowing thirty, forty, and one hundred grains, are recorded.

(*d*) **Mercuric oxide, Red precipitate, HgO.**—This in its usual form is a red powder, slightly soluble in water. Heated strongly, it decomposes, with evolution of oxygen. When prepared by precipitation it is yellow in colour, and in this form is contained in the Lotio hydrargyri flava B.P. Cases of poisoning by it are rare. A fatal case from swallowing an ounce, and a case of recovery after swallowing half an ounce, have been reported.

(*e*) **Mercuric sulphide, Cinnabar, Vermilion HgS.—Hingula** (*Mar.*) **Hingool, Rasa sindura** or **Shingarf.** This is met with either as a dark red crystalline mass, or as a bright red powder. It is only soluble in nitro-muriatic acid, and is entirely volatilised by heat. No acute case of poisoning by it in man has been met with, but cases of chronic poisoning have occurred from its use as a colouring matter for vulcanite plates supporting artificial teeth. A case of chronic poisoning from its over-use as a fumigant is referred to by Taylor.

(*f*) **Mercuric methide, $Hg(CH_3)_2$.**—In two cases of slow poisoning by inhalation of mercuric methide vapour, in addition to salivation, there was impairment of the special senses, of motor power, and of the cerebral functions generally. In one of the two death took place by coma. In the other the patient became idiotic, and after remaining in this state for a year, died of pneumonia.

3. **Mercurous compounds.**—Insoluble or sparingly soluble mercurous compounds are much less actively irritant than similar mercuric compounds. Mercurous compounds are, however, prone to become converted into mercuric compounds. Some mercurous compounds, *e.g.*, the oxide and iodide, undergo this change spontaneously, or under the influence of light only. Others, *e.g.*, calomel, are more stable, but this even is liable under certain conditions to become converted in the body, into mercuric chloride. Of the sparingly soluble or insoluble mercurous compounds, the only one of medico-legal interest is—

Mercurous chloride, Calomel, Hg_2Cl_2.—Ruskapoor. Calomel occurs as a heavy, white, crystalline mass or amorphous powder, almost tasteless, and insoluble in water, alcohol and ether. It is entirely volatilised by heat, and is blackened by solution of ammonia. According to Chevers,[1] the ruskapoor of the Indian bazaars is calomel containing 10 per cent. of corrosive sublimate. In all the specimens I have met with, however, the quantity of corrosive sublimate present has been much smaller. An ordinary medicinal dose of calomel is two to five grains. In large doses it acts as an irritant poison, and in exceptional cases fatal sali-

[1] Med. Jurisp., p. 833.

vation has been caused by ordinary medicinal doses. At the temperature of the body calomel is liable to be converted into corrosive sublimate by solutions of sodium or potassium chloride, and specially by solutions of ammonium salts. In at least two cases (see *Cases* B 27 *a* and *b*) death appears to have resulted from this transformation.

4. **Metallic mercury.—Para, Rasa or Rasam.** Liquid metallic mercury, when swallowed even in large quantity (one to two pounds), seldom produces any ill-effect. In exceptional cases, however (see *Cases* B 28 *a* and *b*), symptoms of chronic mercurial poisoning have been produced. A popular belief appears to exist in some parts of India, to the effect that liquid mercury, when swallowed, causes injury to health. It was administered with this object in case B 28 *b* (see also B 28 *c* and *d*). Metallic mercury in vapour, or in fine division, readily acts on the system; hence workmen in mercury mines, barometer makers, mercurial gilders and platers, and others who are constantly exposed to mercury vapour, are liable to suffer from chronic mercurial poisoning, especially from mercurial tremors. The action of mercury in fine division is very similar to that of an insoluble mercurous salt, and in fine division it may be absorbed through the unbroken skin, and cause chronic poisoning. Mercury in fine division forms one-third by weight of Pilula hydrargyri B.P. and I.P. (blue pill), and Hydrargyrum cum creta B.P. and I.P. The finely divided mercury contained in the latter preparation, is, in hot climates, liable to undergo conversion into mercuric oxide, the preparation as a consequence becoming poisonous.

It may be remarked that in case B 28 *b* the question arose, Is liquid mercury a poison? On this point, Taylor[1] says: "Although liquid mercury is not in itself poisonous, it is liable to be converted into poisonous compounds in the body." Cases B 28 *a* and *b*, however, show that liquid mercury sometimes acts as a poison.

Detection of Mercury.

Elimination of absorbed mercury takes place mainly by the urine and saliva, and may be rapid or slow. The first is most likely to occur in cases of acute poisoning by a soluble mercurial salt, such as corrosive sublimate. Mercury was found to be completely absent from the viscera in a fatal case of poisoning by corrosive sublimate lasting only four days; and Taylor agrees with Orfila in considering that, if in acute poisoning by corrosive sublimate the individual survives fifteen days, it is probable that no mercury will be found in the body. Hence death may occur from mercurial poisoning, and analysis fail to detect the presence of mercury. On the other hand, elimination

[1] On Poisons, p. 360.

of mercury occurs in some cases very slowly, and mercurial preparations are often used in the treatment of diseases. Hence the discovery of mercury in small quantity in the viscera of a deceased individual, is quite consistent with the supposition of death from causes other than mercurial poisoning.

Solid mercurial compounds are most readily identified by reduction. The compound is mixed with powdered sodic carbonate, or powdered sodic carbonate and powdered charcoal, introduced into a test-tube and heated, when globules of metallic mercury sublime. In solution—

Mercuric salts[1] give (1) a yellow precipitate with potassic hydrate; (2) a white precipitate with solution of ammonia; (3) a scarlet precipitate with potassium iodide soluble in excess; (4) no precipitate with hydrochloric acid; (5) a white precipitate, subsequently becoming grey or black, with stannous chloride; and (6) with sulphuretted hydrogen, a precipitate at first white, subsequently orange, and lastly black.

Mercurous salts give (1) a black precipitate with potassic hydrate, or solution of ammonia; (2) a white precipitate with hydrochloric acid or chlorides, blackened by ammonia; (3) a black precipitate with sulphuretted hydrogen; (4) with potassium iodide solution, a green precipitate, soon changing to grey if excess be added.

In organic mixtures mercury may be detected by **Reinsch's process.** On heating the coated copper, a sublimate of globules of metallic mercury is obtained. Or the matters having been boiled with dilute hydrochloric acid (1 to 4), the solution may be treated by the **galvanic deposition process,** as for antimony, using gold foil in place of platinum foil. The coated foil is then heated in a tube as in Reinsch's process.

Quantitative estimation of mercury may be effected by precipitating it as **sulphide.** Corrosive sublimate present in sufficient quantity in organic mixtures, may be separated therefrom by exhaustion with ether.

[1] If the salt under examination is mercuric cyanide, it should be decomposed by HCl before other reagents are added.

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CHAPTER XII.

METALLIC IRRITANTS (*concluded*).

Sub-class II.—Less Volatile Metals.

ZINC, Zn = 65.

CASES of poisoning by this metal are usually accidental, from swallowing either the sulphate or the chloride. Of these the first is a non-corrosive, and the second a corrosive irritant. Besides their local action, zinc compounds, when absorbed, exert a remote specific action on the nervous system, causing great prostration of strength, collapse, convulsions, and, in some cases, impairment of special sensation, *e.g.*, of smell, sight, and taste.

Zinc sulphate, White vitriol, $ZnSO_4$.—Safed tutiya.[1] This is a white crystalline, freely soluble salt, the crystals of which closely resemble in appearance those of magnesium sulphate.[2] In a few instances zinc sulphate has been used criminally, but, as a rule, cases of poisoning by it are accidental, and arise from its being mistaken for magnesium sulphate. When swallowed, it rapidly causes free vomiting, leading to complete, or almost complete, ejection of the poison; hence fatal cases are rare. **Symptoms.**—The usual symptoms of non-corrosive irritant poisoning, with cramps, convulsions, and great prostration of strength. **Post-mortem appearances.**—Those of irritant poisoning. **Dose.**—The least quantity likely to prove fatal cannot be stated with certainty. Half an ounce has caused death, but a dose of two ounces has been recovered from. As an emetic, zinc sulphate is given, in doses of ten to thirty grains, but it should be borne in mind that zinc sulphate is a poison. Hence if, as is sometimes the case in narcotic poisoning, vomiting is not produced, repeated doses are to be avoided (see p. 231). **Acetate of zinc** appears to act similarly to the sulphate, and may be used instead of it as an emetic.

Zinc chloride, $ZnCl_2$.—This is a white, very soluble, and very deliquescent salt, easily fusible, and in the solid condition often met with in cylindrical sticks. A strong solution of it—over 200 grains per ounce—is sold as a disinfectant, under the name of Sir William Burnett's disinfecting fluid. Cases of poisoning

[1] *I.e.*, White metallic salt. [2] And also those of oxalic acid.

by zinc chloride are usually accidental, and most commonly arise from swallowing Burnett's fluid. A case, however, is recorded where death resulted from the application, by a quack, of zinc chloride as a caustic to a cancerous breast. **Symptoms.**—When swallowed in concentrated solution, as is usually the case, the symptoms are those of corrosive poisoning, followed, unless death occurs rapidly, by nervous symptoms, *e.g.*, muscular weakness, tetanic convulsions, impairment of sight, &c. **Post-mortem appearances.**—The lining membrane of the mouth and throat may be found bleached and white, or abraded and inflamed. The gastric mucous membrane has been found grey and corrugated, or inflamed, and in places destroyed; in one case the stomach was found perforated in two places. Where life has been prolonged, contractions of the gullet and stomach have been found. **Dose.**—Severe symptoms have been produced by twelve grains of the chloride. Half an ounce of Burnett's fluid has caused death, but recovery has taken place from doses of one to one and a half ounces.

Treatment of Zinc Poisoning.—The usual treatment for corrosive or non-corrosive irritant poisoning, according to the case. Carbonate of soda should be given as an antidote. Albuminous fluids may also be administered.

Detection.—Solutions of zinc salts (1) acidulated with HCl, give no precipitate with sulphuretted hydrogen; (2) give a white precipitate with ammonium sulphide; (3) give a white precipitate with ammonia solution, soluble in excess; (4) give a white precipitate with potassic hydrate solution, soluble in excess, and forming a solution from which sulphuretted hydrogen throws down a white precipitate; (5) if a solution of a zinc salt be precipitated with sodic carbonate, the solution boiled, and the precipitate collected and ignited with a little cobalt nitrate solution in a platinum dish, the residue in the dish becomes bright green. **From organic mixtures,** zinc may be separated by burning away the organic matter. The ash may be dissolved in dilute acid, zinc obtained from the solution as sulphide, the sulphide dissolved in a little nitric acid, and the solution treated as in (5) above.

COPPER, Cu = 63·5.

All the salts of this metal are non-corrosive irritants.

Acute copper poisoning most commonly arises from swallowing the sulphate or the subacetate. In India attempts at homicide, by the administration of the sulphate in food or sweetmeat, are not uncommon, but the strong metallic taste of this and other soluble copper salts, prevents their being used homicidally to any great extent. Acute—usually mild—accidental cases, from the contamination of food by copper cooking vessels (subacetate), are also tolerably frequent in India. Suicidal cases, and cases

where the poison has been taken with intent to cause abortion, are occasionally met with in England; and a fatal accidental case in a child, from sucking pieces of the sulphate, has been reported.

Symptoms.—A strong metallic taste in the mouth, followed by violent vomiting, and the usual symptoms of non-corrosive irritant poisoning. The vomited matters are usually bluish or greenish, becoming deep blue on addition of ammonia (coloration due to bile is unaffected by ammonia). In severe cases these irritant symptoms may be followed by convulsions, paralysis, and insensibility. In severe cases, also, suppression of urine is common; and jaundice, not present in arsenical or mercurial poisoning, is a tolerably constant symptom.

Fatal cases are rare.—One ounce of the sulphate has caused, and probably less might cause, death, but doses of more than an ounce have been recovered from. Death has occurred (in the case of the child mentioned above) in four hours. In adult cases the fatal period is usually about three days.

Post-mortem appearances.—Those usual in non-corrosive irritant poisoning, plus a yellow tinge of the body, and a blue or green colour, deepened by ammonia, of the contents of the stomach and intestines. Perforation of the intestines has been observed.

Treatment.—The usual treatment for non-corrosive irritant poisoning, with the administration, as an antidote, of albumen or albuminous fluids.

Chronic copper poisoning.—This has been met with, but is not common, among workers in metallic copper and its salts. It is also reported to have arisen from the use of plates as supports for artificial teeth, made of gold largely alloyed with copper. More frequently chronic copper poisoning has been traced or attributed to the contamination of articles of food with copper. The presence of copper, however, in articles of food habitually consumed, provided the quantity present be minute only, does not seem to give rise commonly to chronic poisoning. **Symptoms.**—At first lassitude, giddiness and headache, loss of appetite, and a constant metallic taste in the mouth. Afterwards, great muscular debility, a constant desire to vomit, and diarrhœa, with colicky pains. A purple or green line may be present on the gums, and in some cases the hair is said to have acquired a greenish tint. Acute colic, with constipation and local paralysis (symptoms of chronic lead poisoning), are absent in chronic copper poisoning. **Treatment.**—General, as indicated by the symptoms, and immediate removal from the toxic influence.

Contamination of articles of food with copper.—Copper salts have been used to colour pickles, preserved fruits and vegetables, and confectionery. Sulphate of copper also has been

added to bread, in order, it is said, to promote the fermentation of the dough, and make the bread whiter. Articles of food are very liable to become accidentally contaminated when prepared or kept in copper vessels. On metallic copper (1) pure water has no action if the copper be clean and air is excluded, but if the water is boiled in contact with the copper, and air be present, solution takes place; (2) the solvent action of water on copper is increased when saline matters, especially ammonium salts and chlorides, are present in solution therein; (3) acid and fatty food materials, boiled and allowed to cool, even in perfectly clean copper vessels, take up copper, but acid food materials, boiled in clean copper vessels and poured out at once, do not dissolve the metal; (4) in all cases the liability to contamination is greater if the copper vessel used is dirty. Copper cooking vessels are frequently tinned inside for protection; the tin used should be free from lead, otherwise chronic lead poisoning may result.

Metallic copper, except in very fine division, may be regarded as inert. Cases of chronic poisoning among coppersmiths, from constantly handling metallic copper, have been reported in England; and also cases among workmen using, as in certain printing processes, copper in a state of very fine division.

Sulphate of copper, Blue vitriol, $CuSO_4,5H_2O$.—Moratut, or Nilatutia, readily obtainable in India, occurs in blue efflorescent crystals, which, when heated, lose water and fall into a colourless powder. The salt is very soluble in water, its solution responding to the tests for copper and combined sulphuric acid. Medicinally it is given internally in one-quarter to two-grain doses, as an astringent, and in five- to ten-grain doses as an emetic. For fatal dose and criminal use in human poisoning in India, see *ante*. A few cases of its use in India, as a cattle-poison, have been met with.

Subacetate of copper, Artificial verdigris.—Zangal or Pitra. Several subacetates of copper exist, all compounds of normal cupric acetate $Cu\,2(C_2,H_3,O_2)$ and cupric oxide. They are blue or green in colour, partly soluble in water; are used as pigments, and appear to be as poisonous as the sulphate. Half an ounce of the subacetate has proved fatal to an adult. Food contaminated with copper, derived from vessels in which it has been prepared or kept, commonly contains copper either as subacetate, or as carbonate (natural verdigris). The subacetate, prepared by boiling or steeping metallic copper in an acetous organic fluid, is a common popular emetic remedy in India in cases of poisoning.

Other copper salts.—Green verditer and blue verditer, both oxycarbonates, and Brunswick green, an oxychloride of copper, all used as pigments, are poisonous. So also are the arsenite and aceto-arsenite (see p. 153). The symptoms produced

by these last two compounds, however, are those of arsenical poisoning.

Detection.—Copper in minute quantity is nearly always present in the human liver and kidneys, and in the liver and kidneys of domestic animals. Traces of copper have been detected also in wheat and barley, and in a large number of vegetables. Articles of food, again, frequently contain copper in minute quantity as an accidental impurity.[1] Hence the detection of copper in minute quantity in human viscera, is quite consistent with death from a cause other than copper poisoning. **Solutions containing copper** give (1) a dark brown, almost black precipitate, with sulphuretted hydrogen; (2) a blue precipitate with ammonia, dissolving in excess, with formation of a deep blue or purple solution; (3) a blue precipitate, with potassic hydrate, insoluble in excess; (4) a chocolate brown precipitate with potassium ferrocyanide; and (5) when slightly acidulated with sulphuric acid, deposit metallic copper on a clean iron wire. **Organic matters containing copper** may be incinerated, the ash treated with nitric acid, again incinerated, and the residue dissolved in dilute hydrochloric acid; the solution is then filtered and tested for copper as above. Or for quantitative estimation, the solution may be poured into a weighed platinum dish, a piece of metallic zinc added: this dissolves, the copper deposits on the dish as metallic copper, and after washing and drying, may be weighed in this form.

LEAD, Pb = 207.

Lead poisoning may be acute or chronic. In acute cases the symptoms are those of non-corrosive irritant poisoning, except that there is constipation, not diarrhœa. In chronic cases, much more frequently met with than acute cases, the characteristic symptoms are colic and local paralysis.

Acute lead poisoning. Symptoms.—When a soluble compound, *e.g.*, the acetate, is swallowed, a burning pain in the mouth and throat comes on soon after swallowing the poison, followed by vomiting, and afterwards by colic with constipation. The fæces, if any be passed, are black. Cramps of the flexors follow, and there may be paralysis of the extensors, and a blue line on the gums as in chronic poisoning. Giddiness, stupor, and even coma have been observed. Sparingly soluble compounds cause similar effects, except that the first symptoms do not appear until some hours after swallowing the poison. Fatal cases are rare. **Post-mortem appearances.**—More or less redness of the mucous membrane of the alimentary canal may be found, but this is not always present. **Treatment.**—Promote vomiting or use

[1] Traces of copper are frequently present in native liquor. I found traces of copper in about 80 per cent. of a large number of samples, purchased in different districts of the Bombay Presidency.

the stomach-pump, and then give sulphate of magnesia as an antidote. Subsequently opium may be required to relieve pain, and purgatives to overcome constipation.

Chronic lead poisoning may arise from swallowing, inhaling, or external application, of lead or its compounds. Hence it is met with in those whose occupation exposes them to constant contact with lead or lead compounds; and is also met with as the result of wilful or accidental contamination of articles of human consumption or use, with compounds of lead. In chronic lead poisoning; lead colic, or lead palsy, one or both, may be present.

Lead colic, painter's colic, or colica pictonum.—In this the prominent symptoms are:—At first indigestion, constipation and feeling of depression, with loss of appetite, thirst, a metallic taste in the mouth, and fœtor of the breath. Afterwards there is pain about the umbilicus, usually relieved by pressure, hard obstinate constipation, and quick shallow respiration. The urine is scanty, there is a blue line on the gums, and sometimes delirium at night. Vomiting is a common symptom, but febrile disturbance is rare.

Lead palsy.—This may be the first to appear, or it may follow after one or more attacks of lead colic. It usually commences in the extensor muscles of the hand and forearm, causing **wrist-drop.** Afterwards the muscles of the lower extremities may become affected, and even the muscles of the trunk. As in lead colic, there is a blue line on the gums.

The blue line on the gums is believed to be due to a deposition of lead sulphide in the capillaries, and is rarely absent in chronic lead poisoning. A similar line has, however, been observed in chronic poisoning by other metals, *e.g.*, mercury and silver.

Other effects of lead on the system are (1) it checks the elimination of uric acid, predisposing, therefore, to gout; (2) insanity in some cases, it is believed, is traceable to chronic lead poisoning; and (3) in pregnant females lead poisoning predisposes to miscarriage.

The chief occupations exposing to risk of lead poisoning are,—lead miners, smelters, and refiners; plumbers, pipe layers, printers, and type founders. Lapidaries, barmen, and fishmongers also suffer; the first from handling masses of lead in which precious stones are embedded while being cut; the second from constantly handling pewter pots, and the third from contact with wet, lead-covered surfaces, on which fish are commonly exposed for sale. Again, cupellers, makers of white and red lead and other lead compounds, painters and dyers and others using lead pigments, flint glass makers, and potters using lead glaze, are all liable.

Chronic lead poisoning in non-workers in lead is most frequently due to accidental contamination of drinking-water

with lead. It may, however, arise from accidental contamination or wilful adulteration of matters other than drinking-water.

Contamination of drinking-water.—Pure water has no action on lead if air be excluded; but if air be present, lead hydroxide, slightly soluble in water, is formed. This, by the action of carbon dioxide, becomes converted into a basic carbonate of lead, insoluble in water, but soluble in solution of carbonic acid. The solvent action of water on lead is favoured by the presence of ammonium salts, especially ammonium nitrate. On the other hand, sulphates, phosphates, and carbonates retard or prevent the action. Hence drinking-waters, free, or nearly free, from ordinary saline impurities, are specially liable to contamination from lead pipes, lead-lined cisterns, &c.

Accidental contamination of other matters.—This may arise from the article having been made, or from its being preserved, in vessels made of or soldered with lead, or in earthenware vessels glazed with lead glaze, or other vessels painted inside with a lead paint. Sometimes the contamination is mechanical in character, assisted, perhaps, by oxidation of the lead: *e.g.*, chronic lead poisoning has arisen from the use of flour, ground with stones filled in with lead; and from the use of farinaceous foods, or snuff, or tobacco, wrapped in lead foil. In other cases, the contamination arises from a solvent action exerted by the article, such action being specially liable to be exerted by (1) fatty and saccharine matters, and (2) acid matters (except those acid from the presence of sulphuric acid). Thus, milk kept in lead or lead glazed pans, sugar made in lead vessels, and soup kept in lead-soldered tins, are all liable to contamination. Again, chronic lead poisoning has arisen from drinking cider and beer conveyed in lead pipes; from drinking wine contained in bottles in which shot, used for cleaning them, have been carelessly left; from eating pickles contained in lead-capped jars; and from drinking new rum, contaminated with lead from the lead worm of the distilling apparatus. Old rum, however, is generally free from lead, owing to its precipitation as an insoluble compound, by tannic acid contained in the wood of the casks in which the rum is kept.

Wilful adulteration, &c.—Acetate of lead has been added to cheap wine in order to sweeten it. Red lead and chromate of lead have been used to adulterate snuff, and to colour articles of confectionery. Chronic lead poisoning has arisen from the use of hair dyes, cosmetics, and lotions, containing lead; and from the external application of white lead as a dressing, to a scalded surface.

Chronic lead poisoning is occasionally met with in India as the result of the contamination of drinking-water, and also as the result of the administration of oxide of lead in quack medicines.

Treatment and prophylaxis.—In chronic lead poisoning the first indication of treatment is immediate removal from the toxic influence. In the case of non-workers in lead, the discovery of the toxic influence is frequently a matter of difficulty, necessitating the analysis of all matters habitually used by the patient. Colic may be treated by a combination of purgatives and anodynes: *e.g.*, Epsom salts and senna followed by opiates. In both lead colic and lead palsy either soluble sulphates or iodide of potassium may be given, or the two may be combined, each dose of iodide of potassium being followed after an interval of two hours by a small dose of sulphate of magnesia. Workers in lead should be recommended extreme cleanliness, the least possible contact, and the use as a drink of very dilute sulphuric acid. Every precaution also should be taken to remove or keep down lead dust in the workrooms. The following processes have been recommended for the protection of drinking-water conveyed in lead pipes: (1) Lining the pipes with tin, and (2) keeping the pipes filled for some time with water containing sulphates, or with a solution of an alkaline sulphide. Where, however, a water is liable to contamination, the use of lead pipes, lead-lined cisterns, &c., should be entirely avoided.

Metallic lead is generally regarded as powerless to cause acute poisoning. A case, however, is reported of semi-acute lead poisoning from swallowing small shot, and another where death resulted from accidentally swallowing a quantity of melted lead, the lead in this case acting as a mechanical irritant. Chronic cases due to the action of metallic lead are often met with.

Soluble lead salts.—(1) **Acetate of lead, Sugar of lead, $Pb2(C_2H_3O_2)$.** This is a white crystalline salt, very soluble in water, sparingly soluble in alcohol, and insoluble in ether; heated, it chars, yielding no sublimate. It is not very poisonous, and has been given in divided doses to the extent of eighteen grains, or even more, daily for a week or ten days without ill effect. The same quantity in minute doses spread over a longer period would be far more likely to cause serious symptoms. One ounce has often caused acute poisoning. Fatal cases are rare.

(2) **Subacetate, Goulard's extract, $Pb2(C_2H_3O_2)2PbO$,** is generally met with in strong solution, formed by digesting strong solution of the normal acetate with PbO. The solution is frequently milky, owing to the action of atmospheric carbon dioxide. Its action is similar to that of the acetate.

(3) **Nitrate of lead, $Pb\ 2NO_3$,** a white crystalline salt, very soluble in water, but insoluble in alcohol, is probably as poisonous as the acetate.

Sparingly soluble or insoluble lead compounds. (1) **Monoxide of lead, Litharge, Massicot, PbO.—Murdasang,** yellow or reddish yellow in colour, is slightly soluble in water.

Two tablespoonfuls of it have been swallowed without ill effects. Chevers, however, mentions two cases of chronic poisoning, arising from its use by quacks in India, as a cure for syphilis. In one, serious symptoms came on after swallowing twenty-five grains daily (mixed with white sugar) for five days. In the other, two sowars suffered from lead colic, after swallowing on each of three successive days, one hundred and twenty grains of litharge mixed with sixty grains of "*bans lochan*" (tabasheer). In Europe, litharge has often given rise to chronic poisoning, frequently indirectly, from the solvent action of acetic, fatty, or other acids upon it.

(2) **Red lead, Minium, Pb_3O_4.—Sindura.** The toxic action of this is similar to that of litharge. Taylor mentions a case where a woman recovered after swallowing $2\frac{1}{4}$ ounces. In this case no symptoms appeared for nine hours. Red lead (in the Bombay Presidency, and possibly in other parts of India also) not infrequently forms an ingredient of the paste used for arming abortion-sticks. In a case recently tried in Bombay, a woman, a professed abortionist, convicted of causing miscarriage, was found to have in her possession a number of sticks so armed. Red lead, alone or mixed with arsenious oxide, is sometimes in India employed as a cattle-poison (see *Table XV.*, 6, *Appendix*, p. 504).

(3) **White lead** or carbonate of lead, $PbCO_3$, containing a variable quantity of lead hydroxide; is insoluble in water, but soluble in dilute acids. One fatal case of acute poisoning by it in a child æt. five, is mentioned by Taylor. In this case, although no urgent symptoms were present for three days, the child died in ninety hours.

(4) **Chloride of lead, $PbCl_2$,** slightly soluble in cold, more soluble in boiling water; has, in one case—non-fatal—caused acute poisoning. An oxychloride is also met with, used as a paint, under the name of Turner's yellow.

(5) **Chromate of lead, $PbCrO_4$,** a yellow insoluble salt, used as a paint under the name of chrome yellow; has, in one or two instances—owing to its having been used to colour confectionery—caused fatal acute poisoning. As already mentioned (see p. 154), I have found it used to colour the sweetmeats known as "*til-gul*."

(6) **Sulphate of lead, $PbSO_4$,** a white insoluble salt; is said to be inert, but Woodman and Tidy remark that this is doubtful, as "cases are recorded of sempstresses being poisoned by sucking thread mixed with sulphate of lead, for the purpose of increasing its weight."

(7) **Sulphide of lead, Galena, PbS.**—This, like sulphide of antimony, is sold in India under the name of *Surma*, for use as a collyrium. Owing to its insolubility, it is probably either inert, or only very slightly active. No case of poisoning by it appears to have been recorded.

Detection.—Lead-salts in solution give (1) (except the solution be very weak), with hydrochloric acid, a white precipitate not dissolved or blackened by ammonia, but soluble in boiling water; (2) with sulphuretted hydrogen in slightly acidulated solutions, or with ammonium sulphide in neutral or alkaline solutions, a black precipitate; (3) with potassic hydrate, a white precipitate soluble in excess; (4) with potassium iodide, a yellow precipitate, soluble in boiling-water, and crystallising out on cooling in minute silky crystals, and (5), a yellow precipitate with potassium chromate. Insoluble lead compounds (the sulphide and sulphate excepted) are readily dissolved by nitric acid. The sulphide is only partly dissolved by boiling with nitric acid, the remainder being converted into sulphate. The sulphate may be identified by boiling it with carbonate of ammonia solution: this converts it into carbonate, which, after separation and washing, may be dissolved in acetic acid, and the solution tested for lead. **From organic mixtures** lead may be recovered by incineration; the ash, if treated with sulphuric acid, and again incinerated, yields sulphate of lead, which may be decomposed and brought into solution as above.

Other Metals.

Tin.—The only compounds of this metal of medico-legal interest are stannous chloride, $Sn\,Cl_2$, and stannic chloride, $Sn\,Cl_4$. Solid crystalline hydrates of these salts may be met with, but more commonly the salts are met with in strong acid solution. They are used as mordants in dyeing and are active irritants. Cases of poisoning by them are rare. Solution of carbonate of ammonia, and albumen, are indicated as antidotes.

Bismuth.—Two cases of poisoning by the subnitrate of bismuth, $Bi\,NO_3 2(HO)$, are on record. In one an adult died in nine days from ʒii.; in the other recovery took place from a dose of ʒvi. In both cases the symptoms were irritant. In the fatal case, suppression of urine, salivation, and delirium were present, and well marked post-mortem appearances of irritation were found. Subnitrate of bismuth, Taylor remarks, frequently contains arsenic acid (see also footnote, p. 158).

Silver.—The only salt of this metal of toxicological importance is the nitrate, $Ag\,NO_3$. This is a powerful corrosive irritant, and has in two or three cases caused death. Accidental poisoning by this salt sometimes arises from a portion of a stick slipping down the throat while being used as a caustic. Common salt is the best antidote. In chronic poisoning by silver, a blue line appears on the gums, and the whole surface of the body in time assumes a greyish blue or black colour. (For cyanide of silver, see Cyanides, p. 237.)

Chromium.—Chromic acid, $H_2\,Cr\,O_4$, is a powerful corrosive. Cases of chromium poisoning in man have been almost entirely due to the bichromate, red chromate, or dichromate of potassium, $K_2\,CrO_4\,CrO_3$. This salt is a corrosive irritant, not, however, so corrosive as chromic acid, and in addition has a remote specific effect on the nervous system, causing cramps, dilated pupils, and insensibility. ʒii. has caused death in four hours; and in another case a man, while drawing off a solution of the salt with a syphon, accidentally swallowed a small quantity, and died in five hours. Workmen engaged in the manufacture of potassium dichromate, are liable to suffer from chronic poisoning. The prominent symptoms of this are a bitter taste in the mouth, increased flow of saliva, and violent catarrh, terminating in ulceration and destruction of the

nasal septum. Eruptions on the skin, followed by deep and intractable ulcers, are also present. These symptoms are not observed in workmen engaged in making the neutral or yellow chromate, K_2CrO_4. This, however, when swallowed appears to act similarly to the dichromate, except that it is non-corrosive. Probably other soluble dichromates and chromates act similarly to the potassium salts. The antidote indicated in the treatment of acute poisoning, is magnesia or its carbonate.

Iron.—The only two compounds of this metal of medico-legal interest are ferrous sulphate and ferric chloride. Both are irritants, and have been administered in Europe for the purpose of causing abortion. Ferrous sulphate, green vitriol, *hira kasis* or *hira tutiya, bala dokta*, $FeSO_4$, has in a few cases been used homicidally. It is not a very active poison. One ounce has been recovered from, but severe symptoms were present. Ferric chloride, Fe_2Cl_6, is generally met with in the form of the pharmacopœial tincture, which contains per 100 volumes, 5 parts by weight of iron, equal to about 14½ parts by weight of Fe_2Cl_6. Large doses of the tincture—as much as three ounces—have been recovered from, but smaller doses have caused severe symptoms, and in one case death was caused in five weeks by an ounce and a half.

Barium.—The soluble salts of this metal, and the carbonate (insoluble in water, but soluble in the acids of the stomach) are active non-corrosive compound irritants. When swallowed they cause the usual symptoms of irritant poisoning, with giddiness, cramps, and convulsions. In some cases paralysis and dilated pupils have been observed. Cases have been reported of poisoning by the chloride, nitrate, acetate, and carbonate; this last occurs native as the mineral, witherite, and is used as a rat poison. The smallest doses reported to have destroyed life are sixty grains of the carbonate, and a hundred grains of the chloride, but half a teacupful of the carbonate has been recovered from, and the chloride has been administered medicinally in gradually increasing doses, until the quantity daily administered, in divided doses, has reached forty-eight grains or more, without ill-effects being produced. The proper antidotal remedy in barium poisoning is a soluble sulphate.

Gold.—Auric chloride, $AuCl_3$, is a powerful corrosive poison, but no case of poisoning by it in man is on record. A fatal case from six grains of the fulminate, in which, in addition to irritant-symptoms, convulsions and salivation were present, has been reported.

Osmium.—The tetroxide of this metal, OsO_4, commonly called osmic acid, a very volatile solid, is an active irritant poison, almost as active as arsenious oxide. A few cases of accidental slow poisoning by this substance, one of them fatal, have been reported.

Cadmium.—One fatal case of poisoning by the chloride of this metal in a boy æt. fourteen is reported.[1] The quantity swallowed appears to have been about 250 grains. Death took place in an hour and a half. The symptoms were very similar to those caused by chloride of zinc.

Experiments on animals have shown that compounds of the following metals also are poisonous:—Strontium, cobalt, nickel, uranium, thallium, platinum, palladium, iridium, and rhodium. Compounds of manganese, and of molybdenum, appear also to be feebly poisonous.

Alkaline and Earthy Salts.

Certain alkaline and earthy salts, poisonous only in large doses, may conveniently be considered under this head. Cases of poisoning have been reported from large doses of the following:—

Sodium chloride, Common salt.—This, in large doses, acts as an irritant poison. Half-a-pound has caused death with symptoms of irritant-poisoning, followed by paralysis.

[1] Indian Med. Gaz., 1866, p. 156.

Potassium nitrate, Nitre, Saltpetre, Sal prunelle, KNO_3, Sorakhara, in doses of an ounce or more, has, in several instances, caused death. In one case, however, recovery took place after swallowing six ounces. When swallowed in poisonous doses, besides acting as an irritant, it acts remotely on the nervous system, causing great prostration of strength, and, in some cases, convulsions and partial paralysis. Suppression of urine also has been observed. In some of the fatal cases, death has occurred rapidly, *e.g.*, in two hours and in three hours.

Potassium chlorate, $KClO_3$, in large doses acts as a poison, giving rise, in children, to irritant symptoms with lividity of the surface and collapse; and, in adults, to nephritis. One ounce may be regarded as a fatal dose for an adult, and two drachms has caused death in children.

Acid potassium sulphate, Bisulphate of potash, Sal polychrest, Sal de duobus, $KHSO_4$, in large doses, acts as an irritant poison; ten drachms has caused death in two hours. The salt, in some countries, is popularly believed to possess the power of causing abortion, and fatal cases have arisen from its employment with this object. It is liable to contain as impurities, sulphate of zinc and arseniate of potash.

Potassium Bitartrate (see Tartaric acid).

Common or potash alum, $KAl\,2SO_4$, $12H_2O$.—Phatki. This also, in large doses, acts as an irritant poison, and has, in one or two cases, caused death. Burnt alum, or alum deprived by heat of its water of crystallization, has a slight caustic action.

Sulphate of magnesia, Epsom Salts, $MgSO_4 7H_2O$.—Christison mentions a remarkable case of poisoning by this salt. A boy, aged ten, was given by his father two ounces of Epsom salts in a teacupful of water as a laxative. The boy died of collapse within an hour: there was no vomiting or purging. Another point of medico-legal interest attaching to this salt, is the close resemblance its crystals bear to those of oxalic acid and sulphate of zinc.

CHAPTER XIII.

IRRITANTS, CLASS IV.—VEGETABLE IRRITANTS.—ALKALOIDAL VEGETABLE IRRITANTS.

A LARGE number of plants yield matters capable of acting as irritant poisons. Some of these are simple irritants, possessing little or no remote specific action on the nervous system. Others are compound irritants, causing, in addition to irritation, cardiac depression, *e.g.*, squills and gloriosa superba; or acting on the brain and spinal cord, *e.g.*, cocculus indicus. A few owe their activity to the presence of an alkaloid or vegetable base, *e.g.*, stavesacre and the veratrums. These may be called alkaloidal irritants. Of the remainder, a few, *e.g.*, cocculus indicus and plumbago zeylanica, contain crystalline active principles, not alkaloids. In the great majority, however, the active principle is oily or resinous in nature. In cases of poisoning by the alkaloidal irritants (and by alkaloids generally) administration is indicated of gallic acid, or tannin or decoctions containing tannin. These form insoluble compounds with alkaloids. Or animal charcoal, which removes alkaloids from solution by adhesion, may be given with a similar object.

Alkaloidal irritants.—The detection of these and other alkaloidal poisons mainly depends on the elimination of the alkaloid, and its recognition by chemical or physiological tests. The elimination of alkaloids from organic mixtures is generally effected by Stas' process or one of its modifications, something as follows:—The substance under examination is digested with alcohol, slightly acidulated with an organic acid, say acetic or tartaric acid. The acid alcoholic tincture is strained off, and evaporated at a low temperature nearly to dryness. To the syrupy residue, water faintly acidulated as above is added, and the liquid filtered. The acid watery filtrate is now neutralized with sodic carbonate, and, after neutralization, shaken with some liquid which will dissolve the alkaloid, but which is not miscible with water, *e.g.*, ether, amylic alcohol, or chloroform. The ether or other solvent used is then separated from the water, allowed to evaporate to dryness, and the residue tested for the presence of an alkaloid. Previous to testing, the residue may require purification. This may be effected by re-dissolving it in ether, amylic

alcohol or chloroform, and shaking the solution so obtained, with water slightly acidulated with sulphuric acid. The acid watery fluid is then separated, neutralized, and treated like the original neutralized filtrate. In testing the residue, or purified residue, the first thing is to ascertain whether or no an alkaloid is present. This may be effected by dissolving a portion in very dilute hydrochloric acid, and testing the solution so obtained, by what may be called the group-tests for the alkaloids. The chief of these are, solution of iodine in potassium iodide, solution of auric chloride, solution of platinic chloride, and mercuric potassic iodide solution.[1] All these give precipitates with alkaloids. Having, by means of these group-tests, ascertained that an alkaloid is present; appropriate tests, chemical or physiological as the case may be, may then be applied to other portions of the residue, for the purpose of identifying the particular alkaloid present. (See also p. 301.)

Non-alkaloidal irritants.—The great majority of these do not contain any substance capable of detection by chemical processes. Hence, many can only be recognised by their botanical or physical characters. A few, however, contain matters separable from organic mixtures by chemical processes, and capable of identification by chemical or physiological tests. When such matter is a glucoside or other crystalline substance, its separation may, in many cases, be effected by a modification of the process for elimination of alkaloids just described. This essentially consists in extracting with immiscible solvents, *e.g.*, ether, benzene, &c., the un-neutralized, instead of the neutralized acid watery filtrate (see detection of picrotoxin and plumbagin, *post*, pp. 216 and 219; and see also p. 301).

It may be noted here that the terminations -ine and -ia are used indifferently to denote alkaloids, *e.g.*, strychnine is also called strychnia. The termination -in denotes a non-alkaloidal substance, *e.g.*, picrotoxin, plumbagin.

Alkaloidal Vegetable Irritants.

Melanthaceæ.

Asagræa officinalis (Veratrum officinale, Sabadilla officinarum, or Schœnocaulon officinale), Sabadilla or Cevadilla. From the fruit and seeds of this plant the poisonous alkaloid, or mixture of alkaloids, used in medicine under the name of veratria or veratrine,[2] is obtained. This is an active poison. The medicinal dose is one-twelfth to one-eighth of a grain. In over-doses it

[1] Prepared by adding potassium iodide solution to solution of mercuric chloride, until the precipitate at first formed is just re-dissolved.

[2] Commercial veratrine has lately been found to consist mainly of two alkaloids, veratrine and cevadine, both powerfully sternutatory.

causes violent sneezing, and the usual symptoms of irritant poisoning, coupled with great depression of the action of the heart and collapse. Veratria also first stimulates and then paralyses the peripheral extremities of the sensory nerves; hence a peculiar pricking sensation followed by numbness is one of the symptoms of poisoning by it. In cases of poisoning by veratria, this pricking and numbing sensation is said to be more felt in the fingers and toes and in the joints, than in the tongue; while in cases of poisoning by aconite, which has a similar effect on the sensory nerves, the pricking and numbing sensation is more felt in the tongue. Sabadilla seeds contain about ·3 per cent. of veratria.

Detection.—Veratria may be extracted from organic mixtures by Stas' process, and recognised by the following special tests: (1) It excites violent sneezing. (2) Strong hydrochloric acid dissolves it without change of colour, but on warming the liquid becomes red. (3) Strong sulphuric acid dissolves it, forming a yellow solution, which gradually changes to orange and finally becomes red; on the addition of bromine water to the sulphuric acid solution, a purple colour is produced.

Veratrum album, or White hellebore, and **Veratrum viride,** American or green hellebore. The rhizomes or root-stocks of these, and of other species of veratrum, cause symptoms similar to those caused by veratria. Twenty grains of white hellebore root has caused death, and probably less would prove fatal. Green hellebore root, officinal B.P. and I.P., is less irritant than white hellebore, and rarely occasions purging; except this, its action is similar to that of white hellebore root. The medicinal dose of green hellebore root is 1 to 2 grains. Formerly, the veratrums were believed to owe their activity to the same principles as sabadilla. Lately, however, it has been found that they contain the alkaloids of sabadilla in small quantity only, and that the alkaloids present in them are chiefly jervine, pseudo-jervine, rubi-jervine, and veratralbine, all non-sternutatory bases. All four give a play of colours with strong sulphuric acid, the two last giving colours very similar to veratria, while jervine and pseudo-jervine give a yellow changing to yellow brown, and after a time to green, the green tint becoming more developed on dilution.

Colchicum autumnale, or Meadow Saffron. The whole of this plant, a native of Europe, is poisonous, owing to the presence of colchicin, a nitrogenous substance classed by some chemists as an alkaloid. The corm and seeds are used in medicine chiefly in the treatment of gout. In over-doses, colchicum causes burning pain in the throat and abdomen, violent vomiting and purging, and the usual symptoms of irritant poisoning, coupled with great collapse. The brain, as a rule, is unaffected. In fatal cases death usually occurs within twenty-four hours. After death the

stomach and intestines are nearly always found inflamed. In exceptional cases however, post-mortem appearances of irritation of the alimentary canal have been altogether absent. Colchicum is seldom used criminally as a poison (see, however, *Case* B 3).

Dose.—The medicinal dose of the powdered corm is 2 to 8 grains. The B.P. and I.P. in addition contain—an extract, and an acetic extract of the corm, dose ½ to 2 grains; also a wine prepared from the corm (strength 1 to 5), and a tincture of the seeds (strength 1 to 8), dose of either 10 to 30 minims. In two cases a quantity of tincture equal to 48 grains of the dried corm caused death. This is the smallest fatal dose on record. More than one case of recovery after swallowing one ounce of the wine has been reported.

Treatment.—The general treatment of irritant poisoning, with free administration of decoctions containing tannin. Stimulants should be given to counteract depression, and the patient kept warm.

Detection.—The fresh corm is pear-shaped, about 2 inches long by 1 inch or rather more in width; brown externally, white, firm, and starchy within. When cut it exudes a milky juice, its taste is bitter and acrid. The seeds are reddish brown externally, white within, spherical, and about one-tenth of an inch in diameter. For the separation of colchicin from organic mixtures, Stas' process may be employed, using chloroform as a solvent. Colchicin differs from alkaloids, in being removable from acidulated watery solution by agitation with chloroform; hence the acid watery filtrate obtained in Stas' process may first be shaken with petroleum ether to remove impurities, and then without neutralization with chloroform. Colchicin, unlike alkaloids also, is not precipitated by mercuric potassic iodide solution. The special chemical test for colchicin is the reddish violet colour produced by the action of strong nitric acid upon it. It does not, like veratria, excite sneezing.

Hermodactyl, Surinjan.—Under the name of surinjan two kinds of hermodactyl are sold in the bazaars of India, viz., súrinján-i-shírin or tasteless hermodactyl, and súrinján-i-talkh or bitter hermodactyl. Both varieties of hermodactyl are corms, more or less resembling colchicum corms. Of the two the tasteless variety appears to be nearly inert, while the bitter variety acts like colchicum. The bitter variety has lately been ascertained to be the corm of Colchicum luteum, growing in Cashmere; and the sweet variety, the corm of Merendera persica. Dymock[1] notes that the sliced bulb of the true narcissus (N. Tazetta) is sold in Bombay as bitter surinjan, but that it may be detected by its larger size and tunicated structure. Its action is similar to that of other species of narcissus (see Amaryllideæ, *post*, p. 213).

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[1] Mat. Medica, p. 837.

LEGUMINOSÆ.

Cytisus Laburnum (Laburnum).—All parts of this plant, common in Europe, are poisonous. Several cases of poisoning by it, mostly accidental, have occurred in England. The usual symptoms of laburnum poisoning are vomiting and purging, followed by drowsiness and insensibility, with muscular twitchings and dilated pupils. Its active principle is the alkaloid cytisine. Cytisine is said to be the active constituent of Persian and Australian insect powder.

Cytisus Scoparius (Spartium Scoparium, or common broom). The tops of this plant, also a native of Europe, are officinal in the B.P. and I.P. In large doses they cause vomiting and purging. They contain a crystalline non-poisonous substance scoparin, and the poisonous liquid volatile alkaloid sparteine, the action of which is identical with that of conia, a similar alkaloid contained in conium maculatum, which see (p. 283).

RANUNCULACEÆ.

Delphinium Staphysagria (Stavesacre).—The seeds of this plant have an acrid taste, and from experiments on animals have been ascertained to act as a compound irritant poison, their special remote action being to cause cardiac depression, and paralysis of the respiratory movements. They contain several alkaloids, of which the two most important are staphisagrine, which paralyses the motor nerves like curare; and delphinine, which appears to act very similarly to aconitine, paralysing both the spinal cord and the heart. For other poisons belonging to this order, see non alkaloidal irritants, p. 211, and Aconite, p. 285.

CONIFERÆ.

Taxus baccata, or Yew.—Several cases of poisoning by this plant have occurred in England, most of which have ended fatally. The majority of the cases have been accidental, from children eating the berries or leaves, or from a decoction of the leaves having been given as a cure for worms. In a few cases death has resulted from swallowing a decoction of the leaves with intent to procure abortion. The prominent symptoms are vomiting followed by narcotism, with, in some cases, convulsions and dilated pupils. The respiration is slowed, and death usually occurs by asphyxia, due to paralysis of the respiratory muscles. The active principle appears to be an alkaloid taxine. Yew leaves are sold in the Indian bazaars under the name of *birmi* and *taliopatr*. They resemble savin leaves, except that they have a lancet-shaped apex (see Savin, p. 221).

Other Irritant Alkaloids.

Emetine.—This is the alkaloidal active principle of ipecacuanha, and is an irritant to the gastric mucous membrane, rapidly causing vomiting; it is also a cardiac depressant.

Apomorphine.—This is an artificial alkaloid, prepared by heating morphine with hydrochloric acid. It is the most active emetic known; one-tenth to one-fifth of a grain hypodermically injected, rapidly causes free vomiting, and may be employed to excite vomiting in cases of poisoning.

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CHAPTER XIV.

NON-ALKALOIDAL VEGETABLE IRRITANTS—ANACARDIACEÆ, ASCLEPIADEÆ, CONVOLVULACEÆ, AND CUCURBITACEÆ.

ANACARDIACEÆ.

Marking-nuts, Bhela, Bhilawa (*Hind.*), **Bibba** (*Bomb.*), **Shen-kottai, Sheran-kottai** (*Tam.*), the fruit of the Semecarpus Anacardium. These when bruised yield a brown, highly acrid, oily juice, turning black on exposure to the air. This juice, when applied to the skin, vesicates strongly, raising black blisters, containing a fluid which causes an eczematous eruption on any part of the skin it comes into contact with.[1] Internally administered, the juice appears to be much less actively irritant than it is when externally applied. According to Dymock,[2] the juice of one nut mixed with a seer of milk is an ordinary internally administered dose in native medical practice; and Mahomedan writers speak of 12 to 24 grains of the juice, given in oil or melted butter, as an ordinary medicinal dose, and of 2 dirhems (= 96 grains) as a poisonous dose. Marking-nuts appear to be seldom, if ever, in India, given internally as a poison. The bruised nut, however, is used as a local irritant application for the purpose of procuring abortion; and the juice, like vitriol in England, is thrown over the body to cause injury. A case of this last kind terminating fatally, where marking-nut juice mixed with other irritants was employed, is recorded by Chevers.[3] Again, in a case recently tried before the High Court, Bombay, a Hindoo was convicted of causing hurt to his wife, by throwing marking-nut juice over her face; blistering of the skin and severe ophthalmia of one eye, lasting several days, being the result. Chevers, quoting Honigberger, also mentions the employment of marking-nut juice by malingerers for the purpose of producing ophthalmia and skin eruptions; and Dr. W. Gray once met with a case where a man introduced three marking-nuts into his wife's vagina, apparently

[1] Basiner, quoted by Dymock, Mat. Med., 2nd ed., p. 204.

[2] *Op. cit.*, p. 203.

[3] The other ingredients of the preparation were "nerium odorum, plumbago zeylanica, sulphate of copper, beetles (genus Mylabris), and a snake."

as a punishment for infidelity. The juice, more or less diluted, is said to be used as an application to the skin, for the purpose of imitating bruises in support of a false charge.[1]

Identification.—Marking-nuts in the dry condition in which they are usually met with, are black in colour and more or less heart-shaped, with a rough projection at the base (see Fig. 9). They measure, excluding this projection, in longest diameter from about 7 to 11-tenths of an inch, and weigh from about 25 to 55 grains each. They have a thick cellular pericarp, the cells of which contain the irritant juice. Inside the pericarp is a large flat non-acrid kernel. An acrid juice similar to that found in the pericarp is contained in the thick root bark of the tree.

The acrid juice is soluble in alcohol, ether, and oils. It contains anacardic acid and cardol. Although both these substances are readily decomposed by heat, irritant effects have been produced by exposure to the vapour of the juice. Anacardic acid may be extracted from the juice as an insoluble lead salt, by digesting an alcoholic solution with oxide of lead. Water added to the

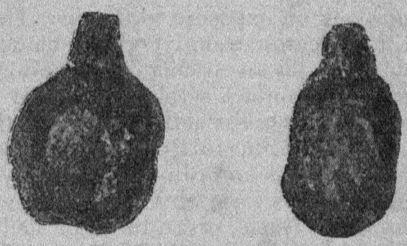

FIG. 9.—MARKING NUTS.

alcoholic solution after removal of the anacardic acid, causes separation of the cardol. Cardol is a yellow oily liquid, insoluble in water, but soluble in alcohol and ether. It blisters the skin strongly, and, according to Basiner, when subcutaneously injected in large doses, causes, in warm-blooded animals, stupor and paralysis. Marking-nut juice may be recognised by (1) its solubility in alcohol, ether, and oils; (2) its vesicating action on the skin; and (3) if a few drops of an alcoholic solution of the juice are placed in a porcelain dish, and a drop of solution of potassic hydrate is added thereto, a bright green colour is at once produced, which, on rolling the fluid about in the dish, rapidly changes to reddish brown. When applied to the skin it should be diluted with oil, and used with caution. When applied diluted it may be some time before it begins to act. Testing in this way some of the fluid used in the Bombay case mentioned above, no effect was noticed for forty-eight hours, after which a painful and very persistent eruption was produced.

[1] Dymock, *op. cit.*, p. 203.

Cashew nuts, Kaju (*Hind.*, *Mar.*, *Bomb.*), **Kottai-mundiri** (*Tam.*), **Hijli-badam** (*Beng.*); the fruit of **Anacardium occidentale.** The pericarp of the cashew nut contains a brown acrid juice of similar composition and properties to marking-nut juice. Its alcoholic solution, however, treated with potassic hydrate solution, turns reddish-brown, and not bright green, like an alcoholic solution of marking-nut juice. Cashew-nut juice appears to be seldom, if ever, used criminally in India. The kernel of the nut is non-acrid, and is eaten raw, or more usually slightly roasted, or cooked.

Rhus.—Some species of this genus of Anacardiaceæ yield a milky juice possessing irritant properties, very similar to those of marking-nut juice. Exposure simply to the vapour of this juice is said to cause distressing symptoms. Woodman and Tidy[1] mention as poisonous—Rhus radicans, poison oak or poison ivy; Rhus vernix, poison sumach; and Rhus toxicodendron. Among the plants of Sind, Murray[2] mentions, as yielding a similar acrid juice, Rhus acuminata (syn. Rhus succedanea, *Linn.*), Tatree (*Hind.*), Arkkol rikul (*Punj.*).

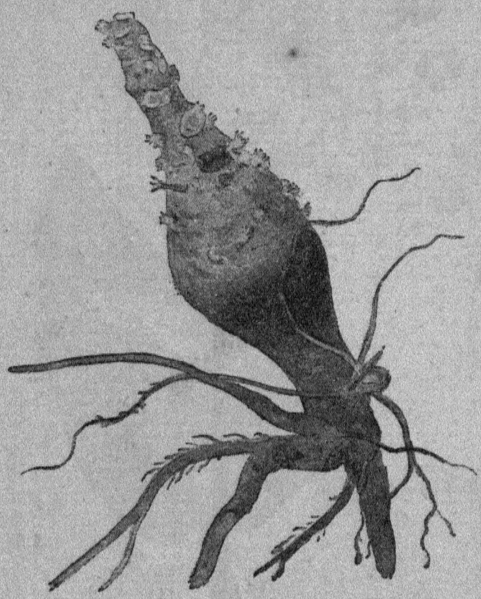

FIG. 10*a*.—CALOTROPIS GIGANTEA—ROOT.

ASCLEPIADEÆ.

Calotropis gigantea and Calotropis procera. Ak, Madar (*Hind.*), **Akanda** (*Beng.*), **Akra, Rui** (*Bomb.*), **Erukku, Erukam** (*Tam.*). These two shrubs closely resemble one another, and are known by the same vernacular names. One or other of them is found growing wild almost everywhere in India. The leaves and stalks, when incised, yield an acrid milky juice, used in

[1] Med. Jurisp., p. 282. [2] Plants of Sind, p. 86.

native medicine as an external application in cutaneous affections and as a depilatory. Harvey[1] states that he has seen severe conjunctivitis set up by the accidental admission of a drop into the eye. The fresh or dried juice, or the root bark, is also given internally as an alterative or purgative. An ordinary medicinal dose of the powdered root bark as an alterative is 3 to 10 grains three times a day. In doses of 30 to 60 grains the root bark acts as an emetic, and has been used as a substitute for ipecacuanha.

FIG. 10*b*.—CALOTROPIS GIGANTEA—STEM, LEAF, AND FLOWER.

In India madar juice appears to be used criminally, chiefly for purposes of infanticide, and as an abortifacient. Harvey, however, mentions a case where a female is reported to have committed suicide by drinking madar juice; and another case where a woman attempted to poison her husband, by introducing madar leaves into a mess of cooked vegetables she had prepared for him. According to Chevers and others, forcing madar juice down the

[1] Bengal Medico-legal Report, p. 72.

throat is a method of infanticide employed by the castes among which female infanticide prevails. Madar juice is also given internally, and applied locally, for the purpose of causing abortion. The active principle appears from a recent investigation by Warden [1] to be a yellow bitter resin, besides which the root-bark also contains two substances named by Warden, madar-alban and madar-fluavil, closely resembling the alban and fluavil found in gutta-percha.

Identification.—The root-bark (officinal I.P.) occurs in short flat or arched pieces $\frac{1}{8}$th to $\frac{1}{5}$th of an inch thick. The outer surface is yellowish grey, soft and corky, fissured lengthwise, and can be easily separated from the middle cortical layer, which is white, mealy, and traversed by narrow brown liber rays. The taste is mucilaginous, bitter and acrid, and the odour peculiar (Dymock). Figs. 10 *a* and *b* show the general appearance of the root, leaves, flowers, &c., of C. gigantea. Drury describes the flowers of C. gigantea as rose-colour and purple mixed, and those of C. procera as pale purple.

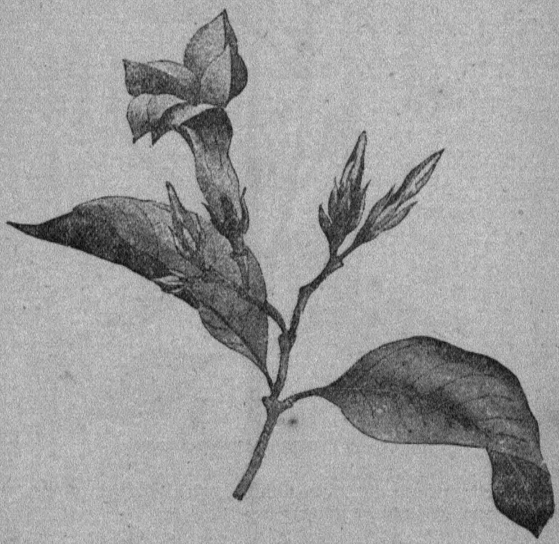

FIG. 11.—CRYPTOSTEGIA GRANDIFLORA.

Cryptostegia grandiflora, *syn.* **Nerium grandiflorum** (see Fig. 11).—This is a climbing plant belonging to the N.O. Asclepiadeæ. The stalk yields a milky juice, which when dried solidifies into a substance resembling india-rubber. One fatal case of poisoning by this plant has been reported. The pounded leaves,

[1] Pharm. Journ., August 22, 1885.

mixed with water, were swallowed. Persistent vomiting came on half an hour afterwards, and the patient—a male adult—died in fifteen hours, apparently from exhaustion. There was no purging, and no head symptoms were present.[1] No alkaloid could be found in the leaves.

Tylophora fasciculata, *vern.* **Bhui-doree** (see Fig. 12).—This plant is abundant in the Southern Concan, where it is used as a rat-poison. One homicidal case of poisoning by administration of the pounded roots in food, has been reported (see *Case* B 29). From the symptoms stated to have been present in this case, viz., tingling in the mouth, dryness of the throat, giddiness, loss of

FIG. 12.—TYLOPHORA FASCICULATA.

power over the extremities, and insensibility, with dilated pupils, the poison appears to be narcotico-irritant in its action.

CONVOLVULACEÆ.

Scammony, the dried milky juice of the Convolvulus Scammonia, is an ash grey gum resin, in rough cinder-like irregular fragments, with a black shining splintery resinous fracture. It is used in medicine, in doses of 3 to 8 grains, as a

[1] Bombay Analyser's Report for 1877–78, p. 20.

drastic purgative. No case of poisoning by it in the human subject is recorded, but in large doses scammony would no doubt act as an irritant poison. Its active principle is a resin named jalapin, identical with the resinous active principle of one variety of jalap.

Jalap.—The dried tuberous roots of the Ipomœa purga (*syn.* Exogonium purga), and other species of Ipomœa; is similar in action to scammony. Both the powdered root, and a resin extracted from it by rectified spirit, are used in medicine, the first in doses of 10 to 30 grains, and the second in doses of 2 to 5 grains. One variety (from Ipomœa orizabensis) contains jalapin; another contains convolvulin, a resin acting similarly to jalapin, but differing from it, in being insoluble in ether, in which jalapin is soluble.

Kala-dana seeds.—Kálá-dánah, Mirchai (*Hind.*, *Beng.*, and *Bomb.*), Kodi kakkatan-virai (*Tam.*); the seeds of Ipomœa hederacea v. cœrulea (*syn.* Pharbitis Nil). These are used in India as a substitute for jalap, the medicinal dose being 30 to 50 grains of the powdered seeds. The active principle is a resin considered by Fluckiger and Hanbury to be identical with convolvulin. No case of poisoning by these seeds has been reported, but in large doses the powdered seeds would doubtless give rise to symptoms of irritant poisoning.

Identification.—Dymock gives the following description of the seeds:—"The seeds resemble in shape those of most of the convolvuli, being in the form of a segment of a sphere; they are generally about $\frac{3}{16}$ths of an inch in length, and nearly as much in breadth, but sometimes much smaller. Their weight varies from ½ to nearly 1 grain; the colour of the testa is black, except at the umbilicus, where it is brown. Upon soaking the seeds in water the testa bursts and discloses the delicate albumen which envelopes the folded cotyledons and radicle. These have an acrid taste and earthy odour." The same author notices that in Bombay the seeds of Ipomœa muricata are more common than those of the true kaladanah. These are similar in action and appearance to true kaladanah seeds, except that they are larger and heavier, weighing about 3 grains each, and are rather lighter in colour.

Ipomœa Turpethum, Nisot, Nakpatar, Pithori (*Hind.*), Nishotar, Phukari (*Bomb.*), Shivadi (*Tam.*), Teori (*Beng.*).—The root of this plant, common throughout India, is used in native practice as a purgative. It appears to contain a resin similar to jalapin and convolvulin. The medicinal dose of the powdered root is from fifteen to thirty grains.

Cuscuta reflexa, Akasweli, Ghagar bel (see Fig. 13).—This is a parasitic plant with white bell-shaped flowers, common on bushes in some parts of India. According to Assistant-Surgeon Dulip Singh,[1] the *dhais* in the Punjaub have

[1] Ind. Med. Gazette, January 1885.

great faith in a decoction of this plant as an abortifacient. A decoction of 180 grains of the plant, made with boiling water, suffices, it is stated, to produce depression with nausea and vomiting, followed by abortion.

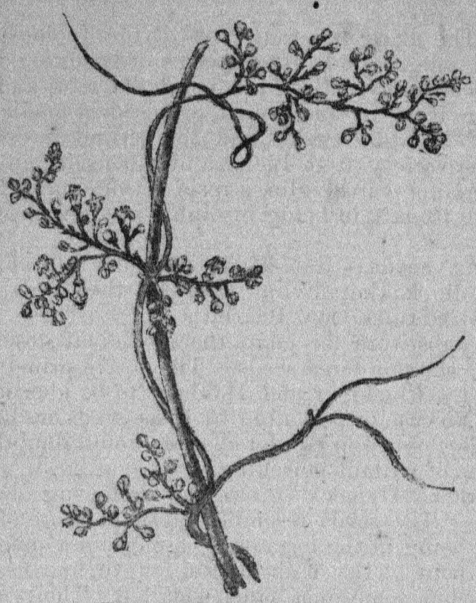

FIG. 13.—CUSCUTA REFLEXA.

CUCURBITACEÆ.

Elaterium.—This is the sediment from the expressed juice of the Ecbalium Elaterium (*syn.* Ecbalium officinarum), or squirting cucumber. It is a powerful cathartic, and is used in medicine as a purgative in doses of one-sixteenth to half a grain. One grain has caused severe symptoms, and probably but little more would be required to cause death. The active principle is elaterin, a white crystalline substance obtainable by adding ether to a chloroform solution of elaterium. Elaterin is soluble in alcohol, and not precipitated from its alcoholic solution by tannin.[1] Dymock notices that the fruit of this plant, or of a closely allied species, is sold in the Bombay shops under the name of Kateri-indráyan.[2]

Identification.—The fruit is 1½ to 2¼ inches long, oblong ovoid, pale yellowish green, and covered with numerous short fleshy prickles, terminating in white elongated points. When ripe it

[1] Pharmacographia, p. 262. [2] Mat. Med., p. 352.

separates suddenly from the stalk, violently expelling the juice and seeds.

Bryonia dioica, or white bryony.—This is a common hedge plant in England, climbing by its tendrils on bushes, &c. It has small faint green flowers, and red clustered berries, each containing six seeds. The root is spindle-shaped, fleshy, and pale, and was formerly used in medicine as a purgative. A decoction of the root has caused death, and children have been accidentally poisoned by eating the berries. The active principle is bryonin, apparently a glucoside.

Citrullus Colocynthis.—Indrayan (*Hind.*), Peykomatti, Tumati (*Tam.*), Kururundawan (*Mar.*). The colocynth of the pharmacopœias, used in medicine as a purgative in doses of from 2 to 8 grains, is the dried and powdered fruit pulp of this plant. In large doses it acts as an irritant poison, and has in Europe in several instances caused death. Christison[1] mentions a case where a teaspoonful and a half of the powder (about 90 grains) proved fatal, and Taylor[2] cites the case of an adult female who took 120 grains of the powder in order to cause abortion, and died in fifty hours. A case of recovery from a dose of 3 ounces is, however, reported. The active principle is a glucoside, colocynthin. In India both the fruit pulp and the root are used in native medicine as purgatives.

Identification.—The dried fruit of Indian colocynth is yellow-brown in colour, and about the size and shape of an orange. It contains a scanty greyish-white pulp, in which are a number of brown seeds.[3] "The root is fibrous, tough, and stringy, and of a yellowish-white colour. All parts of the plant are very bitter, and the dust when dry very irritating to the eyes and nostrils" (Dymock).

Other Cucurbitaceæ.—Many other plants of this order possess properties more or less resembling those of the three just mentioned. Some, it is true, yield edible fruits, but "there is reason to believe that some if not all the edible sorts of cucurbits owe their freedom from poisonous properties to cultivation, for some in a wild state are found to possess them in much activity."[4] The principal Indian species mentioned by different writers as being either in common use as purgatives, or as having given rise to cases of poisoning, are as follows:—

Lagenaria vulgaris.—The bottle-gourd, Tumbi, Karwi-tumbi (*Hind.*), Tiktalau (*Beng.*), Karwa-bopla (*Bomb.*), Shora-kai (*Tam.*), Hurrea-kuddoo (*Duk*), Anapa kai (*Tel.*).—The fruit of the wild variety of this is bitter

[1] On Poisons, p. 595. [2] On Poisons, p. 522.

[3] These are of flattened ovoid form, measure two-tenths by three-tenths of an inch, and are disposed in vertical rows on three thick parietal placentæ, which project to the centre of the fruit, then divide and turn back, forming two branches directed towards one another (Dymock, p. 337).

[4] Lindley, quoted by Birdwood, Bomb. Veg. Prod., p. 157.

and powerfully emetic and purgative. The wild variety, dried, is made into musical instruments, bottles, &c., and it is stated that some sailors were once poisoned by drinking beer which had been standing in a bottle made from one of these gourds.[1] Dr. Kirtikar informs me that he has met with two cases in which smelling the fruit produced violent sneezing.

Citrullus amarus.—This is the wild form of the C. vulgaris, or water-melon. The fruit is very bitter, and is used medicinally as a purgative. The dried fruits are sold in the bazaars of Sind under the name of "kirbut."[2]

Cucumis sativus.—The common cucumber. The fruit of the wild form of this, also called C. Hardwickii, *vern.* Puhari indrayan, is said to possess properties similar to those of official colocynth.

Cucumis trigonus (*syn.* C. pseudo-colocynthis), Karit (Dymock), Indrayan, Bislumbhi (Phar. of Ind.).—This has bitter fruit of the size and shape of a small egg. The fruit is used as a substitute for colocynth. In 1883 a case was reported to the Bombay Chem. Analyser's Office, in which it was stated that the roots of this plant had been administered for the purpose of procuring abortion. A variety, C. trigonus *var.* pubescens, Takmaki, is much less bitter, and is used as a vegetable.

Trichosanthes dioica, Pulbul, Potole.—Kanny Loll Dey[3] states, with reference to this plant, "The bulbous part of the root is a hydragogue cathartic." This plant is cultivated in Bengal and Guzerat for its fruit, which is used as an article of food. An alcoholic extract of the unripe fruit, in three to five grain doses, is described as a powerful cathartic.[4] T. cucumerina, Kadu-padavala, or Ban-padavala (*Mar.*), has similar medicinal properties, and is the Patola of Bombay; it is not cultivated and the fruit is never eaten. Similar properties appear also to be possessed by T. palmata, Lal-indrayan (*Hind.*), Kaundal (*Bomb.*), Mákál (*Beng.*), Koratti, Shavari-pazham (*Tam.*), the fruit of which, Dr. Kirtikar informs me, is sometimes used as a cattle-poison.

Momordica Cymbalaria (*syn.* Luffa tuberosa), Kadavanchi (*Mar.*) (see Fig. 14).—Dymock, in regard to this, writes, "The whole plant is acrid. The fruit is about 1 inch by 1½ inch, has eight prominent ribs, is covered with silky hairs, and while still green dehisces into four parts discharging its seeds." The roots are tuberous and ovoid. Three cases in the last few years have been reported to the Bombay Chem. Analyser, in which it was stated that abortion had been caused by the administration of a decoction of these roots.

Momordica Charantia, Karela (*Hind.*), Kárlá (*Bomb.*), Pava-kai (*Tam.*) (see Fig. 15).—The fruit of this is bitter, but wholesome. It is eaten, but requires to be steeped in salt water before being cooked. A case was reported to me in 1879, in which it was stated that swallowing a decoction of the roots of this plant caused abortion at the seventh month.[5]

Luffa acutangula, var. amara, Karvi-turai (*Hind.*), Kadú-sirola, Kadu-dorka (*Bomb.*), Ghosha-lata (*Beng.*), Sondu-bir-kai (*Tel.*)—Dymock describes the fruit as smooth, 3 inches to 5 inches long, ovoid, marked with ten prominent sharp longitudinal ridges, and having at the apex a small operculum rather more than ½-inch in diameter, which is deciduous. The seeds are grey, and marked with small, irregular, black, prominent specks. Sakharam Arjun[6] describes the fruit as violently cathartic and emetic.

Luffa echinata, Kukar-wel, Deodangri (see Fig. 16).—The fruit of this is described by Dymock as "oval, about the size of a nutmeg, armed with numerous long rather soft diverging bristles, obscurely divided into three cells by numerous dry fibres, and opening at the top with a perforated stopple, which falls off when the seeds are ripe. Seeds about eighteen (ovate, compressed, black and scabrous),

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[1] Lindley, quoted by Birdwood, Bomb. Veg. Prod., p. 157.
[2] Murray's Plants and Drugs of Sind, p. 41.
[3] Drugs of India, p. 118.
[4] Beng. Disp., p. 351.
[5] Bombay Chem. Analyser's Report, 1879–80.
[6] Bombay Drugs, p. 59.

testa very hard, kernel white." The fruit of this is also stated by Sakharam Arjun to possess purgative properties, and Dr. Kirtikar[1] has reported a fatal case in an adult of poisoning by the fruit. One fruit only was eaten; death took place in nine hours, the symptoms present closely resembling those of cholera.

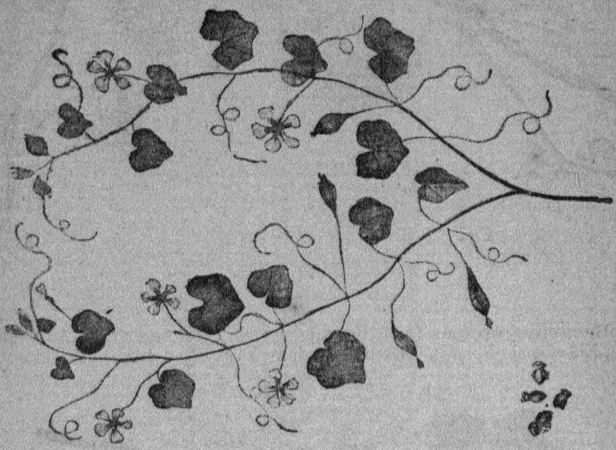

FIG. 14.—MOMORDICA CYMBALARIA.

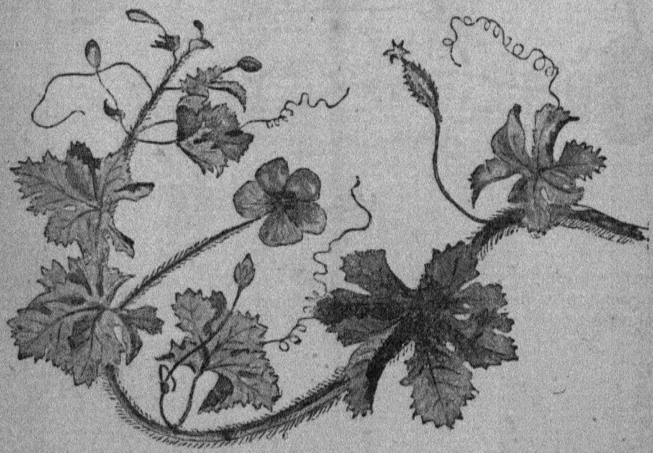

FIG. 15.—MOMORDICA CHARANTIA.

[1] Trans. Bombay Med. and Phys. Soc., 1887.

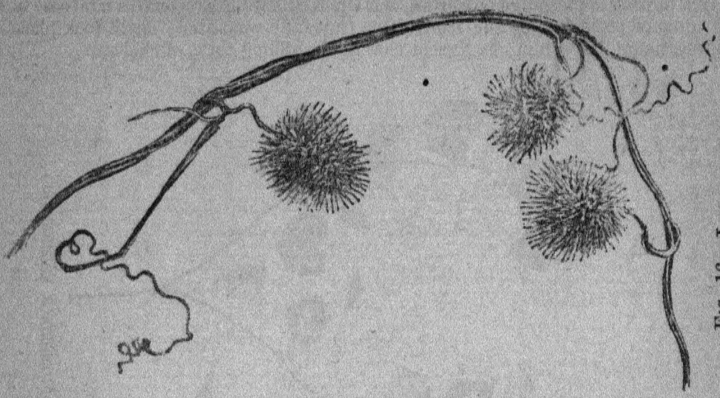

FIG. 16.—LUFFA ECHINATA.

Corollacarpus epigæa (*syn.* Bryonia epigæa), Rakas-gaddah, Akas-gaddah (*Hind.*), Karwi-nai (*Bomb.*), Akasha-garudan (*Tam.*) (see Fig. 17).—The root of

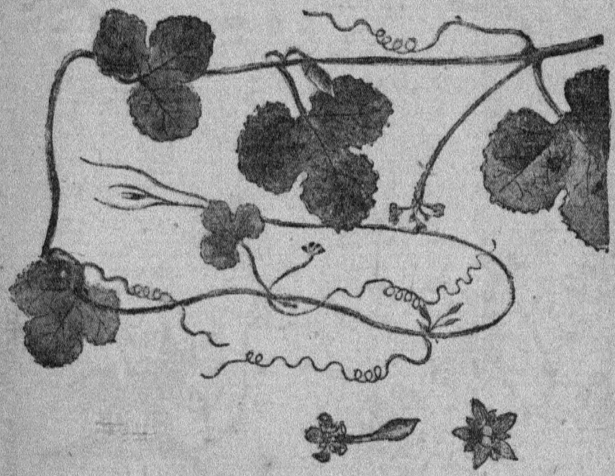

FIG. 17.—COROLLACARPUS EPIGÆA.

this plant contains a yellow bitter principle, which, Dymock suggests, may possibly prove to be identical with bryonin. The root is used, in doses of about one drachm in twenty-four hours, as a purgative.

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CHAPTER XV.

NON-ALKALOIDAL VEGETABLE IRRITANTS — EUPHORBIACEÆ, LILIACEÆ, RANUNCULACEÆ, THYMELACEÆ, AROIDEÆ, AND AMARYLLIDEÆ.

EUPHORBIACEÆ.

Euphorbium and the Euphorbias.

Euphorbium, or Gum Euphorbium, *vern.* Farfiyun, is the dried milky juice obtained by incising the fleshy branches of Euphorbia resinifera, a leafless perennial plant resembling a cactus, growing only in Morocco. It is used in Europe in veterinary practice as a vesicant. Applied to the skin it causes irritation and vesication, and when swallowed is an extremely active irritant poison. A teaspoonful given to an adult in mistake for rhubarb, caused death in three days. Inhalation of the dust raised in grinding euphorbium, is reported to have caused headache, giddiness, and stupor.[1]

Fluckiger found euphorbium to owe its acrid properties to an amorphous neutral resin readily soluble in cold spirits of wine. Of this 38 per cent. was present in the sample examined. In addition, the sample contained 22 per cent. of **euphorbon**, a crystalline substance, sparingly soluble in cold but freely soluble in boiling alcohol, and soluble also in ether. The remaining 50 per cent. was made up of mucilage, malates, and mineral compounds. If euphorbon, deposited from solution in alcohol in a thin film, is moistened with oil of vitriol, and strong nitric acid be slowly added by means of a glass rod, a fine violet hue appears.[2] Lactucerin, contained in Lactucarium, gives, however, the same reaction.

The Euphorbias, a genus of the N. O. Euphorbiaceæ, all yield an acrid milky juice possessing properties similar to those possessed by the juice of E. resinifera, and probably also possessing the same chemical composition.[3] Various writers mention the following Indian species, as plants the juice of which is employed in native practice, externally as a caustic or vesicant,

[1] Christison on Poisons, p. 589. [2] Pharmacographia, p. 504.
[3] Flückiger has also found euphorbon in E. Tirucalli and E. cattimandoo.

or internally as a purge. Occasionally, also, the juice of one of the euphorbias, or a twig of one of them, is used as a local irritant application for the purpose of causing abortion; or homicide is attempted by mixing the juice with food.

Euphorbia Tirucalli, Milk hedge. Thor niwal (*Bomb.*); Kalli (*Tam.*); Tirucalli (*Mal.*); Lunka sij (*Beng.*).—Dymock mentions that 1 to 4 drops of the juice of this are given as a purge.

Euphorbia neriifolia, Sehund, Thohar (*Hind.*), Mansasij (*Beng.*), Newarang, Mingut (*Bomb.*), Ilaik kalli (*Tam.*).—Ainslie, quoted by Dymock, states that the usual dose of the juice of this plant, given by native practitioners as a purge, is about twenty-grains.

FIG. 18.—EUPHORBIA ROTHIANA.

Euphorbia antiquorum, Schadida-calli (*Mal.*), Shadray Kullie (*Tam.*), Bontajammoodoo (*Tel.*), Narashij, Seyard (*Hind.*), Narsij (*Beng.*) (Drury).—Dymock also mentions E. pilulifera, E. thymifolia, and E. parviflora, all three known in the Southern Concan under the name of Nayeti.[1] Drury refers to the use as a vesicant of the fresh juice of E. cattimandoo;[2] and W. Gray[3] mentions a case in which the juice of E. Rothiana (see Fig. 18) was administered internally

[1] Mat. Medica, p. 694. [2] Useful Plants, p. 204.
[3] Bombay Chem. Analyser's Rep., 1874–75.

to a sick man, who shortly afterwards died, but whether from the disease, or the effects of the medicine, could not be made out from the particulars furnished of the case.

Croton Seeds and Oil.

Croton Tiglium.—Jamálgota (*Hind.* and *Bomb.*); Jaypal (*Beng.*): Nervalam (*Tam.*), Naypalum (*Tel.*), Cadelavancu, Neervaula (*Mal.*).—Both the seeds of this plant, and the oil expressed therefrom—croton oil—are highly poisonous. The oil is used in medicine as a purgative in doses of one-third of a minim to one minim. Applied to the skin, it vesicates. The Linimentum crotonis B.P., used as a counter-irritant external application, consists of one volume of croton oil, to seven volumes of a mixture of equal parts of cajuput oil and rectified spirit. Three drops of the oil proved fatal to a child one year old, and half a drachm has caused death in an adult. In one case an adult died in four hours, from a dose of 2½ drachms. One or two grains of the seeds when swallowed, suffice to cause severe pain, with copious watery stools. A case also is reported in which severe symptoms (pain and collapse but no purging) appear to have been produced, by inhaling the dust raised in emptying packages of the seeds.

Kobert, from recent researches, attributes the activity of croton oil to crotonoleic acid (distinct from crotonic acid) present in the oil both free and as a glyceride. Besides this, croton oil also contains the glyceride of a peculiar acid,—tiglic or tiglinic acid,—isomeric with angelic acid, $C_5 H_8 O_2$.

Identification.—The seeds are oval, more arched on the dorsal than on the ventral surface, about half an inch long by nearly two-fifths of an inch broad, and weigh about four grains each. The testa is black, thin, and brittle, and more or less covered by a thin cinnamon brown coat. The kernel is white, is enclosed in a delicate white membrane, and easily splits into two halves, between which lie two foliaceous cotyledons, and a short thick radicle. The oil may be extracted from the seeds or other matters by exhaustion with ether, and recognised by its vesicating action on the skin.

The seeds of the following are stated to resemble those of Croton tiglium in appearance and properties. Baliospermum montanum, *vern.* Danti. These, Dymock states, are often sold as jamálgota by druggists; and Croton oblongifolium, Baragach (*Beng.*), Ganasur (*Bomb.*), Gonsurong (*Goa.*).

Castor-oil Seeds and Oil.

Ricinus communis, the Castor-oil plant.—Arandi (*Hind.*), Erendi (*Bomb.*), Bherenda (*Beng.*), Amanak-kani-chedi Sittamunak, Valluk (*Tam.*), Citavanakoo, Avanak, Pandiavanak

(*Mal.*), Sittamindi, Amidum (*Tel.*).—Castor-oil, if expressed from the peeled and winnowed seeds, without the aid of heat, is mildly purgative. The seeds, however, are highly poisonous. Three grains of the seeds has caused alarming symptoms, and a case is reported where three seeds proved fatal to an adult in forty-six hours. I have met with two cases (one fatal) of poisoning by castor-oil seeds administered in food. According to Stillmark the poisonous principle of the seeds is an albumenoid body (ricin), which gives rise to violent inflammation of the alimentary canal, but not to catharsis. He estimates that ten seeds contain about one-tenth of a grain of ricin, or sufficient to cause death in an adult.

Identification.—Castor seeds resemble croton tiglium seeds in shape and internal structure, but are somewhat smaller. The testa also differs in colour, being grey marked with brown blotches. The oil is distinguished from other fixed oils by being soluble in glacial acetic acid, and in alcohol. It is completely soluble in four volumes of spirits of wine at 15° C. There is also a large variety of castor seed, of a reddish colour with brown blotches, the oil obtained from which is much used for industrial purposes.

Physic Nuts.

Jatropha Curcas.—Jangli arendi (*Hind.*), Bág-bherenda (*Beng.*), Moghli erendi (*Bomb.*), Kattamanakku (*Tam.*), Galamark (*Goa.*).—The fruit of this and of the other jatrophas named below (physic nuts) contain oily poisonous seeds. The action of these seems to be similar to that of croton tiglium seeds, but somewhat milder in degree. The oil expressed from the seeds irritates the skin, and given internally in doses of twelve to fifteen drops is powerfully purgative. Severe vomiting and purging have been caused by swallowing a few grains of the cake left after expression of the oil from the seeds. Several cases of accidental poisoning by physic nuts are recorded, and Chevers mentions one where, in addition to the usual irritant symptoms, muscular twitchings, deafness, impairment of sight, and loss of memory were present. The fruits of **J. multifida** and of **J. glandulifera**, Undarbibi, Jangli erendi (*Bomb.*), Lal-bherenda (*Beng.*), Addaley (*Tam.*), Nela-amida (*Tel.*), are quite as poisonous as those of J. curcas.

Identification.—The fruits of all are three-celled and three-seeded. J. multifida has fruit as large as a walnut of the shape shown in Fig 19. Those of J. curcas are of about the same size, but more uniformly oval in shape, and those of J. glandulifera are not bigger than a hazel-nut, oval, and marked externally with six deep longitudinal grooves. The three plants also may be

distinguished by the shape of their leaves (see Fig. 20). The seeds of all three varieties, in shape and internal structure closely resemble castor-oil seeds. J. curcas seeds are about three-

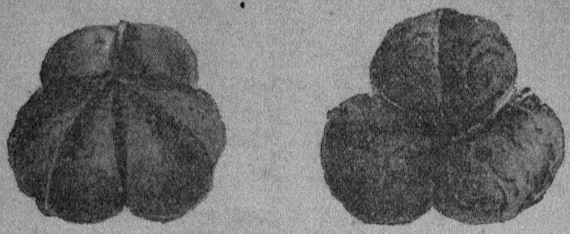

FIG. 19.—PHYSIC NUTS (*Jatropha multifida*).

quarters of an inch long and rather less than half an inch broad. J. multifida seeds are somewhat broader, and J. glandulifera seeds are only three-tenths of an inch long and two-tenths of an

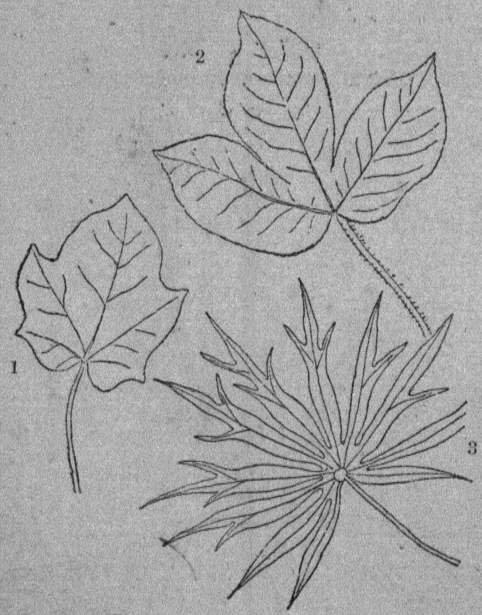

FIG. 20.—JATROPHA LEAVES (1. Curcas; 2. glandulifera; 3. multifida).

inch broad. J. curcas and J. multifida seeds are black with a white scar at one end, while J. glandulifera seeds are grey with two brown stripes on the dorsum.

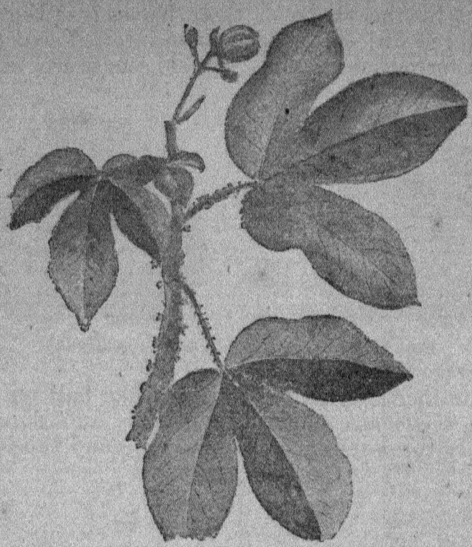

FIG. 21.—JATROPHA GLANDULIFERA.

FIG. 22.—JATROPHA MULTIFIDA.

Other Euphorbiaceæ.

The following other plants of this order are of medico-legal interest:—

Manihot utilissima (*syn.* Jatropha manihot), the cassava plant.—This is a native of the West Indies, where its starchy root is used as an article of food under the name of cassava. From the root also is prepared the tapioca of commerce. Two varieties of it exist—a sweet and a bitter variety. The latter abounds in a poisonous milky juice containing hydrocyanic acid, to which its toxic power is due. Owing to its volatility, however, this is entirely removed by heat, and hence bitter cassava root may be utilised as an article of food, after the juice has been squeezed out and the root has been cooked.

Jatropha urens.—This also is a native of the West Indies, has leaves covered with stinging hairs, merely touching which has, it is said, in two instances caused serious symptoms. In one of the two reported cases extreme collapse came on in a few minutes. In the other "the pain and swelling in the part touched, lasted for some days."[1]

Hippomane Mancinella, Manchineel.—This tree, also a native of the West Indies, yields an acrid milky juice, which, applied to the skin, causes violent inflammation, and if administered internally acts as an irritant poison. Christison mentions that the H. biglandulosa and H. spinosa are equally poisonous.[2]

Hura crepitans, the sand-box tree.—Chevers, on the authority of Dr. H. Cleghorn, remarks that this, introduced from the West Indies, is not uncommon at the Presidency towns. The seeds are violently purgative.[3]

Lebidieropsis orbiculata, Wodisha (*Tel.*), Wodagu marum (*Tam.*) (Drury); Odwan, Nachutu (*Tam.*) (Gribble).—Drury, in regard to this shrub, states, "The bark or outer crust of the capsules is said to be very poisonous;" and Gribble[4] mentions that it has recently been identified as the probable poison, in some cases of irritant poisoning occurring in the Madras Presidency.

LILIACEÆ.

Aloes.—Elwa (*Hind.* and *Beng.*), Elia, Pivalabola, Kalabola (*Bomb.*), Kariya-pólam (*Tam.*).—This well-known medicinal substance is the inspissated juice of the leaves of the Aloe vulgaris and other species of aloe. It is a powerful drastic purgative, acting specially on the rectum. The usual medicinal dose is three to five grains. In large doses aloes is an irritant poison; two drachms has caused death. Aloes has a stimulant action on the uterus, and in Europe is frequently given or taken for the purpose of procuring abortion. A form in which it has often been used for this purpose is hiera picra or holy bitter, a mixture of four parts aloes to one of canella bark. Aloes also is an ingredient of Morrison's, and most quack purgative pills. The active principle of Barbadoes aloes is barbaloin, a glucoside. Other varieties contain nataloin and socaloin, bodies probably members, with barbaloin, of a homologous series.

Squill.—Officinal squill is the sliced and dried bulb of Urginea Scilla (*syn.* Scilla maritima). In doses of six to fifteen grains it

[1] Taylor on Poisons, p. 504.
[2] On Poisons, p. 592.
[3] Med. Jurisp., p. 275.
[4] Med. Jurisp., p. 255.

acts as an emetic, and in larger doses as an irritant poison, causing vomiting, purging, griping pain, strangury, and bloody urine. In poisonous doses squill causes marked depression of the heart's action. Convulsions also have been observed in poisoning by this drug.[1] Twenty-four grains of the powder has caused death. The active principle is scillitin, apparently a glucoside.

Urginea indica.—Kánde, Jangli-piyaj (*Hind.* and *Beng.*), Kol-Kanda, Kochinda (*Bomb.*), Nari vengayam (*Tam.*).—The bulb of this, if young and small, acts similarly to officinal squill, and is largely used in India as a substitute for it.[2] It has, however, been stated that when the bulbs grow old and large they lose their activity. In a case referred to me in 1883, a quantity of food on examination was found to contain portions of bulbs exactly resembling those of Urginea indica. This food, it was stated, had been given by a woman to her husband, who noticed it to possess a bitter taste, and after eating a portion of it suffered from vomiting.

Identification.—Tunicated bulbs (U. indica as large as a common onion, U. scilla larger), each scale made up of polyhedral cells, covered on both sides by an epidermis provided with stomata. The cells are loaded with mucilage, and contain numerous needle-shaped crystals of calcium oxalate.

Gloriosa superba.—Lánguli, Karihári, Kulhári (*Hind.*), Bisha-languli (*Beng.*), Indai, Nagkaria (*Bomb.*), Kalaippaik-kizhaugu (*Tam.*).—The tuberous root of this plant is popularly believed in India to be highly poisonous. According to Moidin Sheriff, however, it is not poisonous in twelve-grain doses, but acts as an alterative tonic and antiperiodic.[3] In large doses it appears to be poisonous, acting in the same manner as squill (see *Case* B 30), and Warden has recently isolated a bitter principle from the root, which he has named superbine, and considers to be closely allied to, if not identical with, scillitin.

Identification.—Dymock describes the root (see Fig. 23) as "tuberous, cylindrical, and flattened, often seven to eight inches long and about one inch in diameter; it consists of two tubers which unite at a right angle, one being much smaller than the other; at the point of union may be seen on the upper surface a circular scar marking the attachment of the stem, and on the under surface, immediately beneath it, another, to which a tuft of thin rootlets is often attached. The tubers are covered with a brown epidermis, except at the point, which is tapering and nearly white, like the growing part of a young kidney potato.

[1] Christison on Poisons, p. 214.

[2] Scilla hyacinthoides (*syn.* Ledebouria hyacinthoides), Paharikanda, Nání-jangli-kándo (*Bomb.*), Shiri-naru-vengayam (*Tam.*), has a scaly bulb about the size and shape of a small pear, which is used as, and appears to be an equally satisfactory substitute for, officinal squill.

[3] Dymock's Mat. Medica, p. 833.

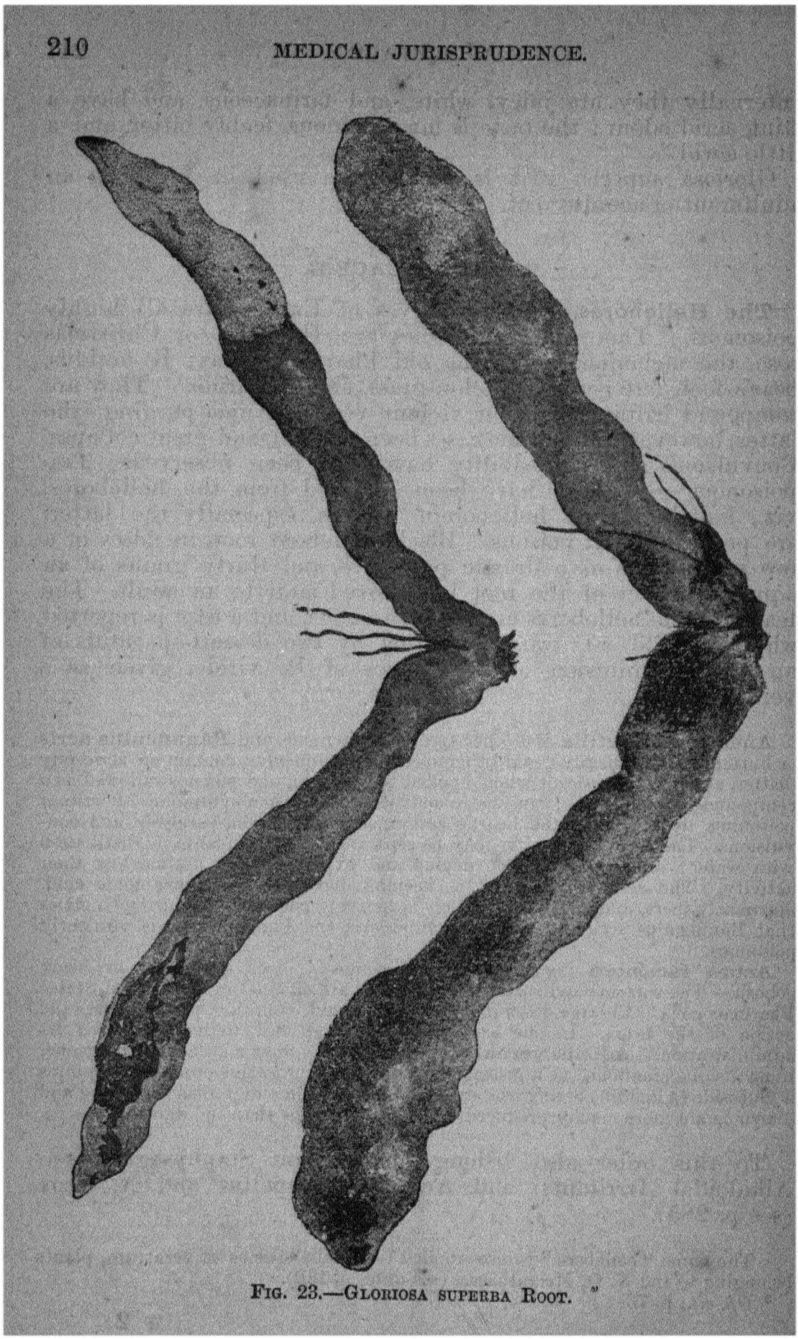

FIG. 23.—GLORIOSA SUPERBA ROOT.

Internally they are juicy, white, and farinaceous, and have a faint acrid odour; the taste is mucilaginous, feebly bitter, and a little acrid."

Gloriosa superba root is said to be used in India as an adulterant of aconite root.

RANUNCULACEÆ.

The Hellebores.—These, natives of Europe, are all highly poisonous. The principal species are H. niger, or Christmas rose, the melampodium of the old Pharmacopœias; H. fœtidus, bear's-foot, fetter-wort, or felon-grass, and H. viride.[1] They are compound irritants, causing violent vomiting and purging (the latter, however, has in some cases been absent), and great collapse. Convulsions and insensibility have also been observed. Two poisonous glucosides have been obtained from the hellebores, viz., helleborin and helleborein. These, especially the latter, are powerful heart poisons. Black hellebore root, in doses of a few grains, acts as a drastic purgative, and thirty grains of an aqueous extract of the root has proved fatal to an adult. The leaves of the hellebores are also poisonous, and a case is reported where a child, æt. two, was killed by two dessert-spoonfuls of an aqueous infusion of the leaves of H. viride, given as a vermifuge.

Anemone pulsatilla and other species of anemone, and **Ranunculus acris** or buttercup, R. sceleratus, and other species of ranunculus, contain an acrid oily matter, acting as a vesicant when applied to the skin, and when swallowed as a compound irritant poison, causing, in addition to the usual symptoms of irritant poisoning, depression of the heart's action, slow respiration, paralysis, and convulsions. The acrid oily matter may be separated from the plants by distillation with water; and the plants, on drying and exposure, after a time lose their activity. The acrid oily matter on keeping decomposes into anemonic acid, apparently inert, and anemonin, which is actively poisonous. Murray[2] notices that Ranunculus sceleratus grows in Sind and the Punjaub, and is virulently poisonous.

Actæa racemosa (*syn.* Cimicifuga racemosa), black snake-root or black cohosh.—The rhizome and rootlets of this plant are officinal in the United States Pharmacopœia. In large doses it causes nausea and vomiting, and depresses the action of the heart. In one case abortion is reported to have followed its administration. **Adonis vernalis,** regarded by some as a species of anemone, must also be mentioned as a poisonous plant belonging to this order. It contains a glucoside (adonidin) acting as a cardiac depressant like digitalis. Vomiting and diarrhœa are more readily produced by Adonis vernalis than by digitalis.

To this order also belong Delphinium Staphysagria (see Alkaloidal Irritants) and Aconitum Napellus and A. ferox (see p. 285).

[1] The name "hellebore" is also applied to certain species of veratrum, plants belonging to the N. O. Melanthaceæ (see *ante*, p. 186).

[2] *Op. cit.*, p. 73.

THYMELACEÆ.

Daphne Mezereum, or Mezereon; **Daphne laureola**, or spurge-laurel, and other species of the same genus, are compound irritant poisons. A few accidental fatal cases of poisoning by the bright red berries of Daphne Mezereum are recorded, the symptoms being vomiting and purging, followed by narcotism with dilated pupils. The bark also of Daphne Mezereum, and of other species of daphne, is poisonous, and appears to contain a fatty vesicating oil. Several members of this genus are found in India, and Burton Brown[1] mentions that the root and stem of one species (apparently D. papyracea), growing on the hills, is a powerful irritant, known under the vernacular name of Gundhera, and that it can be recognized by the peculiar woody tissue which forms the inner bark, and which is composed of long white fibres, easily separable from the wood.

Lasiosiphon speciosus, *vern.* **Rametha.**—The bark of this shrub, common on the Ghauts, is a powerful vesicant, and is used for poisoning fish. Dr. W. Gray mentions a case in which administration of the leaves of the plant caused irritant symptoms, followed by abortion and death.[2] Dymock describes the bark as acrid in taste and consisting "of an outer tuberous portion, which is of a light brown colour, and divided by numerous transverse and longitudinal fissures, so that it can be easily separated, and of an inner layer, which is white, tough, and silky like mezereon."

AROIDEÆ.

The tuberous roots of **many of the arums**, a genus of this order, when taken into the mouth, cause immediate burning pain, great swelling of the tongue, and salivation, and when swallowed give rise to the usual symptoms of irritant poisoning, followed by convulsions, dilated pupils, and insensibility. The roots of many contain much starch, and are used, after washing and baking, as articles of food. Pedler and Warden from a recent investigation, consider the activity of the roots to be due to mechanical irritation set up by the needle-shaped crystals of oxalate of lime with which these roots abound. The following may be specially mentioned:—

Arum maculatum, lords and ladies, cuckoo-pint.—This, common in England, has given rise to several cases of accidental poisoning, chiefly among children, from eating the leaves. The tuberous root—poisonous when fresh—after steeping in water and baking, is used as an article of food under the name of Portland sago.

Arum seguinum—dumb cane.—A native of the West Indies. Two drachms of the juice of this plant has been known to prove fatal in a few hours.

Arum montanum, Konda-rakis (*Tel.*), and **Arum lyratum, Adavi.**—These are both, Drury states, natives of the mountainous parts of the Northern Circars. The root of the first is so poisonous that it is used to poison tigers; the root of the second is used as an article of food, but requires careful cooking.

Synantherias sylvatica (*syn.* **Arum sylvaticum**), **Uzomut** (*Goa*), **Wajrmut** (*Mar.*).—Dymock notices that the seeds of this produce the local effects noted above, followed by numbness, and states that the crushed seeds are used in the S. Concan, on account of their benumbing effect, as a remedy in toothache.

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[1] On 'Poisons used in the Punjaub,' quoted by Chevers, p. 285.
[2] Bombay Analyser's Rep., 1874–75, p. 10.

Arum colocasia (*syn.* Colocasia antiquorum), *vern.* Kachu.—Two cases of poisoning by the tubers of this plant, both non-fatal, were reported to the Chemical Examiner, Bengal, in 1886.

Dr. Kirtikar informs me, also, that he has met with cases in which irritation of the throat followed the eating of the tuberous root of **Amorphophallus campanulatus** (*vern.* Sooran, Madanmast), and the spadix of **Pythonium Wallichianum** (*vern.* Shewla), two plants belonging to this order, the named portions of which are commonly used as vegetables in India.

AMARYLLIDEÆ.

Narcissus pseudo-narcissus, or daffodil.—This, common in England, is mentioned by Guy and others as an irritant poison. Other species of narcissus also have a similar action (see Hermodactyl, p. 187). The root of an Indian plant of this order, viz., Crinum asiaticum, *var.* toxicarium, is officinal in the Pharm of India as an emetic, and Dymock mentions that the bulb of Crinum ornatum, Gadambikanda (*Bomb.*), is extremely acrid, and is used for blistering cattle, a slice being bound on the skin.

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CHAPTER XVI.

NON-ALKALOIDAL VEGETABLE IRRITANTS FROM OTHER ORDERS.

Argemone mexicana, N. O Papaveraceæ; Bharbhand (*Hind.*), Brahma-dandi (*Sans.*), Shiál kánta (*Beng.*), Dáruri (*Bomb.*), (Dymock), Feringie-datura, or Peela-datura (*Duk.*), (Balfour).—The seeds of this plant yield an oil which, when swallowed, causes vomiting and purging. In 1878 four cases of accidental poisoning, from the use of this oil in food, in each of which several persons were affected, were reported to the Bo. Analyser's office.

Identification.—The seeds are contained in prickly capsules, three-quarters to one and a half inches long. They are dark brown in colour, nearly globular, about one-fifth of an inch in diameter, and covered with minute, regularly arranged, projections and depressions. One hundred of them weigh about three grains. The oil, shaken with an equal volume of strong nitric acid, acquires a deep crimson colour, the acid at the same time becoming similarly coloured. The crushed seeds, warmed with strong nitric acid, give a similar reaction. Dragendorff has found the seeds to contain an alkaloid possessing reactions similar to those of morphia.

Capsicum annuum, N. O. Solanaceæ; Lal-mirch (*Hind.*, *Bomb.*, and *Beng.*), Milagáy (*Tam.*).—The fruits of this (chillies), and of other species of capsicum, contain an exceedingly acrid, volatile, non-alkaloidal substance, capsicin, apparently the active principle; and also a volatile alkaloid, with an odour like conia. Applied to the skin, capsicum causes irritation and vesication, and, taken internally in sufficient quantity, acts as an irritant poison. Owing to the volatility of the active principle, the fumes arising from burning capsicum are highly irritant. The medicinal dose is half a grain to one grain.[1] Woodman and Tidy mention a case where quack pills containing capsicum proved poisonous to a woman aged seventy-four; and Taylor[2]

[1] Bentley and Redwood, Mat. Medica, p. 625. Taylor on Poisons, p. 505, gives the medicinal dose as five to ten grains.

[2] On Poisons, p. 505 (*R.* v. *Stevens*, C. C. C., May 1864).

mentions a case where a quack was tried for causing the death of a boy, aged fifteen, who was suffering from hip-joint disease, by giving him a mixture containing Cayenne pepper; the prisoner, however, was acquitted. Capsicum is frequently used in India for purposes of torture. Chevers mentions its use for this purpose in the following ways:—Introduction into the nostrils, eyes, vagina, or urethra; burning it under the nose, rubbing it on the breasts of females, and covering the head with a bag which has contained it.

Identification.—The appearance of the fruit is well known. The seeds are of a flattened kidney shape, about a quarter of an inch long and wide, and closely resemble dhatura seeds. Like dhatura seeds, the testa under the microscope is seen to be covered with convoluted ridges. They differ from dhatura seeds (1) in having a pungent taste; (2) the convex border is single, not double, as in the dhatura seed; and (3) on section the embryo is seen to differ in shape from that of the dhatura seed (see p. 266, and Figs. 33 and 34).

Cocculus indicus, or Levant Nut.—The fruits or berries of the Anamirta cocculus, N. O. Menispermaceæ; *vern.* Kakmari (*Hind.*), Kákphul, Karwi (*Bomb.*), Kákkáy-kolli-virai (*Tam.*).—These are highly poisonous, owing their activity to the presence of picrotoxin, a crystallisable non-alkaloidal substance, contained in the seeds, but not in the pericarp of the fruit. The pericarp, in fact, is non-poisonous, and an entire berry might therefore possibly pass through the body without causing bad symptoms. Picrotoxin is a compound irritant poison, causing vomiting, purging, &c., with extreme giddiness, followed by delirium and epileptiform convulsions, stupor, and loss of voluntary power. A few cases of poisoning by Cocculus indicus berries have occurred in Europe and America. In one case a child died from the application of an alcoholic tincture of the berries to the head. A decoction or extract of cocculus indicus has been used in England as an adulterant of beer, porter, &c., in order to increase its intoxicating power; and Birdwood states that it is said to be largely used for the same purpose by the liquor retailers of Bombay. In England, cocculus indicus has been used by thieves to stupefy their victims in order to facilitate the commission of theft; and in 1881 a case was referred to the Bombay Analyser's Office, in which it was alleged that it had been used for a similar purpose. The Bombay Analyser's records also show that during the last ten years cocculus indicus was detected in three cases of alleged cattle poisoning. Cocculus indicus berries are used in England as a fish poison, and are apparently also sometimes used in India for the same purpose. In medicine, cocculus indicus is only used as an external application as a parasiticide (see also *Spinal Poisons*, p. 281).

Identification.—The dry berries are nearly globular, about half an inch or rather less in diameter, and have a blackish wrinkled surface. On section they are seen to contain a peculiar mushroom-shaped body, consisting of a cup-shaped seed, supported on a stalk formed by a projection inwards of the endocarp (see Fig. 24). The pericarp is tasteless, the seed is bitter. From organic mixtures picrotoxin may be separated by digesting the matter under examination with alcohol, evaporating the alcoholic tincture nearly to dryness, and treating the extract so obtained with acidulated water. The acid watery solution is then to be filtered, shaken with ether, and the latter separated and evaporated to dryness, when picrotoxin, if present, will be found in the residue. Picrotoxin is not precipitated from solution by the group tests for the alkaloids (see p. 185). It has a bitter taste, reduces alkaline copper solutions like grape sugar, and dissolves in strong sulphuric acid, forming a yellow solution, which on addition of a trace of potassium dichromate becomes violet, changing into brown.

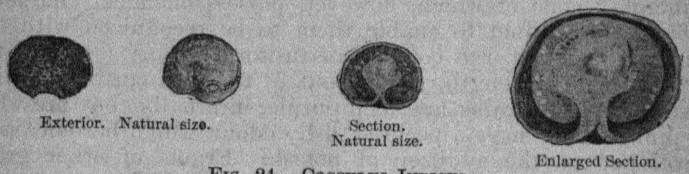

FIG. 24.—COCCULUS INDICUS.

Dolichandrone falcata (ss. Spathodia falcata, and Bignonia spathacea), N.O. Bignoniaceæ, *vern.* Mersinghi.—Dymock notes that "this small tree has the reputation of being used to procure abortion." The bark is also, it is stated, used as a fish poison. I found, however, no ill effects to follow the administration of a considerable quantity of a decoction of the bark to a small dog.

Ergot.—This is the scleroticum (compact mycelium, or spawn B. P.) of Claviceps purpurea, a fungus liable to be developed within the paleæ of numerous plants of the order Graminaceæ. Rye so affected constitutes the ergot of rye used in medicine. Other cereals, *e.g.*, wheat and oats, are liable to the affection.[1] In large single doses ergot causes the usual symptoms of irritant poisoning, accompanied by headache, giddiness and stupor, depressed action of the heart, and dilated pupils. Acute poisoning by ergot is very rarely met with. One case, however, is recorded where a woman in the fourth month of pregnancy, having swallowed a quantity of powdered ergot, aborted and died soon afterwards from peritonitis. Outbreaks of ergotism, or chronic poisoning by ergot, arising from the use of affected grain as food, have occurred in France and Germany. Ergotism shows itself

[1] Chevers mentions that in India a disease called "leera" appears in barley and oats, and a similar disease called "kindol" appears in Bajree, and points out that grain thus affected resembles ergot of rye.

in two forms—a convulsive and a gangrenous form. Both commence with gastro-intestinal disturbance. In the first giddiness, aberrations of vision, loss of sensation in the hands or feet, spasms and convulsions, are the prominent symptoms. In the second, dry gangrene of the extremities is the principal effect. Ergot causes contractions of the uterus, especially of the pregnant uterus, and hence is used sometimes criminally as an abortifacient. In the early stages of pregnancy, however, it sometimes fails to excite contractions of the uterus. Thus Taylor mentions a case where a woman took about a teaspoonful of tincture of ergot three times a day for eleven weeks, and died at about the third month of pregnancy without having aborted.[1] The medicinal dose of powdered ergot in uterine diseases is 5 to 15 grains three times a day, but its administration should not be continued for any length of time. The dose for a woman in labour is 20 to 60 grains. Doses of 30 to 120 grains have caused symptoms of poisoning.

Identification.—Ergot appears to contain more than one active principle,[2] none of which, however, possess chemical characters sufficiently distinct to enable them to be recognized with certainty. Ergot of rye consists of fusiform grains $\frac{1}{4}$ of an inch to $1\frac{1}{2}$ inch in length, and about $\frac{1}{6}$ to $\frac{1}{3}$ of an inch thick. Externally the grains are deep purple, internally they are white or pink. They have a peculiar fishy odour, which becomes more developed on the addition of potash. Ergots of other grains chiefly differ from ergot of rye in length and thickness.

Gamboge, Ussarah-i-Rewand, Gotaganba (*Pers.*, *Hind.*, and *Bomb.*), Mukki (*Tam.*).—This is a gum resin from Garcinia Morella, N. O. Guttiferæ, and is used in medicine in doses of one to five grains as a drastic purgative. One drachm has caused death. A case, however, occurred in Bombay in which a girl æt. nineteen, intending to commit suicide, swallowed three drachms, but recovered under treatment.

Identification.—A tawny or brownish orange substance, generally met with in cylinders 1 to $2\frac{1}{2}$ inches in diameter, with an acrid taste, forming a yellow emulsion with water, and violently purgative.

Moringa pterygosperma (the Horse-radish tree), N. O. Moringeæ, Sahjna (*Hind.*), Shegva, Shegat (*Bomb.*), Murungai (*Tam.*).—The fruit of this tree is eaten as a vegetable, and the root is used as a substitute for horse-radish, which it exactly resembles in taste. Kanny Loll Dey, quoted by Chevers,[3] states that one of the methods of procuring abortion in use near Calcutta, is the administration of a dose consisting of about half an ounce of pounded sahjna bark mixed with twenty-one black pepper-corns, and that this is a very dangerous means, the mother as a rule dying when it is resorted to.

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[1] Taylor's Manual, p. 513.

[2] The latest researches are those of Kobert; according to this authority ergot contains three active principles, viz. (1) ergotinic acid, this paralyses the spinal cord, but does not appear to act on the uterus; (2) sphacelinic acid, this causes spasmodic contraction of the arterioles, resulting in gangrene or extravasations of blood; and (3) cornutin, a convulsant causing clonic and tonic spasms.

[3] Med. Jurisp., p. 716.

Plumbago zeylanica.—Chitrak (*Hind.*), Chita (*Beng.*), Chitra (*Bomb.*), Chittira or Chittira múlam (*Tam.*); and **Plumbago rosea** (*syn.* P. coccinea), Lal-chitrak, chita, or chitra (*Hind.*, &c.), Shivappu chittira (*Tam.*), N. O. Plumbagineæ (see Fig. 25).—The roots, and probably other portions of these plants, contain a highly acrid crystallisable non-alkaloidal principle called plumbagin. The bruised roots applied to the skin cause vesication. Taken internally, in large doses, plumbago root acts as an irritant

FIG. 25.—PLUMBAGO ZEYLANICA.

or narcotico-irritant poison. In India plumbago root is sometimes administered internally as a poison, and Chevers[1] refers to two fatal cases, one of them a case of homicide, in which it was so employed. More commonly in India plumbago root is used for the purpose of causing abortion. With this object it is sometimes given internally. I have more than once detected plumbagin in pills stated to have been administered for this

[1] Med. Jurisp., p. 252.

purpose. Usually, however, it is employed as a local irritant application to the os uteri, a portion of the root or a twig of the plant being pushed into the vagina, and sometimes even into the uterus. In other cases the cotton-covered end of an abortion stick (see p. 376) is smeared with a paste made from the powdered roots; and I once met with a case in which a lump of such paste was simply thrust into the upper part of the vagina, and was found there after death.

Detection.—The roots are ¼ to ½ an inch in diameter, dark brown externally and reddish within; from them and matters containing it, plumbagin may be extracted by digesting the substance under examination with alcohol, straining this off, and evaporating the tincture to dryness. The dry residue from the tincture should then be digested with a small quantity of water rendered slightly alkaline with caustic potash, the solution obtained filtered, acidulated with hydrochloric acid, and shaken with ether. The ether is then separated, evaporated to dryness, and the residue tested for plumbagin. Plumbagin treated with caustic potash solution dissolves, forming a bright crimson liquid. Hydrochloric acid added to this changes the colour to yellow, and on standing the liquid deposits yellow flocculi of plumbagin, which may be separated by shaking the acidified fluid with ether. An alcoholic solution of plumbagin gives a crimson precipitate with solution of basic acetate of lead.[1]

Plumieria acutifolia (*syn.* P. acuminata), N. O. Apocynaceæ; Khairchampa (*Bomb.*), Gobur champa (*Beng.*), Dolochápo (*Guz.*) (see Fig. 26).—This small tree, common in India, has blunt truncate branches and white and yellow flowers. It abounds in viscid juice, which dries into a substance resembling india-rubber. "The root is a violent cathartic, and the blunt-ended branches are used to procure abortion."[2] A case of poisoning by the bark has recently been reported.[3] The quantity taken was about a square inch. This was pounded, mixed with water, and swallowed by a man aged twenty-five as a remedy for colic. The symptoms were vomiting, depressed heart's action, and somewhat dilated pupils. The plant does not give any blue or green reaction with hydrochloric acid, like Cerbera Thevetia and C. Odallum (see p. 299). It contains plumieric acid in combination with calcium.[4]

Randia dumetorum, N. O. Rubiaceæ; Mainphal (*Hind.*), Gelaphal (*Bomb.*), Maruk-kallán-kay (*Tam.*), Menphal (*Beng.*), Mindhal (*Guz.*) (see Fig. 27).—Dr. George Bidie[5] states that the fruit of this is apparently an irritant emetic, and is used to produce emesis by the poorer classes in Mysore, the dose being one ripe fruit well bruised. According to Ainslie, an infusion of the root-bark also acts as an emetic; and Dymock mentions that the bruised nut is used in the Concan to preserve grain from the attacks of insects, and as a fish poison.

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[1] A colouring matter obtained from rhubarb root gives, with caustic potash solution, a crimson colour similar to that given by plumbagin. An alcoholic solution of it, however, gives, with acetate of lead solution, an orange precipitate, not a crimson precipitate like plumbagin.

[2] S. Arjun, Bomb. Drugs, p. 210.

[3] Surgeon K. R. Kirtikar, in Trans. Bo. Med. and Phys. Soc., 1886.

[4] Watts, Dict. viii. 11, p. 1656.

[5] Pharm. of India, p. 118.

Chevers,[1] on the authority of Edgeworth, states that the fruit is used in the neighbourhood of Jullundur, as an ingredient in medicines given by the mouth for the purpose of procuring abortion.

Identification.—Dymock[2] gives the following description of the dried fruit:—"About the size of a crab apple, globular or oval, reddish brown, crowned with the rim of the calyx. It consists of a pericarp and shell, which contains the seeds embedded in pulp. The shell is hard and thick, two-celled, the dividing septum being thin and membranous. The pulp is grey, and has a nauseous taste and smell. The seeds are small and oblong, about $\frac{1}{8}$th of an inch in length, slightly flattened, very hard, and of a brown colour. The

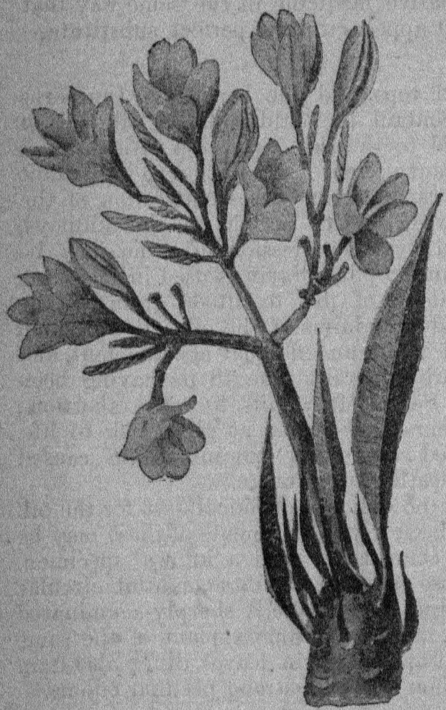

FIG. 26.—PLUMIERIA ACUTIFOLIA.

FIG. 27.—RANDIA DUMETORUM.

average weight of the fruit is about sixty grains." In the dried fruit the seeds are found agglutinated together into two rough masses, each something of the shape of a coffee-bean, but larger, one mass in each cell of the fruit.

Ruta graveolens, Rue, N. O. Rutaceæ, yields by distillation a pale yellow volatile oil of a strong disagreeable odour and

[1] Med. Jurisp., p. 720. [2] Mat. Medica, p. 408.

acrid bitter taste. The oil—officinal B.P. and I.P.—appears to be the active principle of the plant, and is used in medicine, internally as an emmenagogue in doses of from two to five drops, and externally, as a rubefacient. Garrod gives the medicinal dose of the powdered leaves as twenty to forty grains. In large doses, rue acts as an irritant poison, causing vomiting and prostration, with a feeble slow pulse and coldness of the extremities. Swelling of the tongue and salivation have also been observed. In Europe, rue has been frequently given or taken, it is said, with success, as an abortifacient. **Ruta angustifolia**, Sudáb (*Hind.*), Sutáp (*Bomb.*), Arvada (*Tam.*), is cultivated in most parts of India, and is used in native medicine in the same way that R. graveolens—for which it appears to be a perfect substitute—is used in Europe.

Savin.—The leaves and tops, and the berries of Juniperus Sabina, N. O. Coniferæ, contain an acrid volatile oil—oil of savin—which, when applied to the skin, acts as a vesicant, and when swallowed, as an irritant poison. In addition to its irritant action on the alimentary canal, savin causes congestion of the kidneys and uterus. Owing to its action on the kidneys, strangury, hæmaturia, and even suppression of urine, may be present in cases of poisoning by it. Savin is used in medicine as an emmenagogue in doses of five to fifteen grains of the powdered tops, or one to fifteen minims of the oil. In England, savin is popularly believed to be an effectual abortifacient, and cases of poisoning by it are generally due to its having been given with this object. Savin often fails to cause abortion; sometimes it succeeds, generally, however, at great risk to life. Several cases are recorded where its administration caused abortion, followed by the death of the woman.

Identification.—There is no reliable chemical test for the oil. If the leaves or tops have been given, fragments of these may be found and identified by comparison with a known specimen. The woody fibres, like those of other Coniferæ, exhibit circular discs. The leaves are minute and have a sharply-acuminated point, while those of yew, another poisonous plant of the same order, have a lancet-shaped apex. Savin leaves differ also from yew leaves in giving off when rubbed a strong peculiar odour.

Soapnuts, Ritha (*Hind.* and *Bomb.*), Pönnau-kottai (*Tam.*), Ringin (*Mar.*), the fruit of Sapindus trifoliatus (*syn.* S. emarginatus), N. O. Sapindaceæ.—These are used in India as a detergent, and also in native medicine. According to Dymock, the pulp, in four-grain doses, is given internally as an anthelmintic and tonic, and a native authority recommends its administration in seventy-grain doses, with about eight grains of scammony, as a purgative. Soapnuts contain the glucoside saponin, a white amorphous powder, soluble in water, and forming a solution which froths like a solution of soap. Saponin is an undoubted poison. Blyth states that one and a half to three grains of saponin administered by the mouth produces slight symptoms in the human subject, and estimates that forty grains administered subcutaneously to an adult would endanger life.

Saponin locally applied acts as an irritant, subsequently producing paralysis and anæsthesia; absorbed into the system, it paralyses the nerve-centres and the heart. Hence, as stated by Blyth, in poisoning by saponin the symptoms would probably be great muscular prostration, weakness of the heart's action, and diarrhœa, and after death post-mortem appearances of irritant poisoning would probably be found. Saponin is present in many other plants, *e.g.*, in senega; in sarsaparilla; in the bark of Quillaia saponaria, soap bark, and in Agrostemma Githago or common corn-cockle. Blyth suggests that accidental admixture of corn-cockle seeds with wheat, may possibly account for some of the mysterious cases of poisoning which have occurred from time to time after eating cakes, bread, &c.

Identification.—The dry berries of S. trifoliatus have a shrivelled brown translucent skin, and are met with either in threes united together or separate, and showing a heart-shaped scar on one side; each berry contains a single dark red-brown seed of similar shape to the fruit. The fruit-pulp has a fruity smell; its taste is sweet at first, afterwards very bitter. **Saponin** may be obtained from matters containing it by digesting them with hot spirit, from which the saponin deposits as the liquid cools. The deposited saponin may then be purified by dissolving it in the least possible quantity of boiling water. On the addition of absolute alcohol to this, the saponin precipitates. Saponin is insoluble in cold alcohol and in ether, strikes a red colour with sulphuric acid, and dissolves in water, forming a soap-like solution.[1]

Terminalia bellerica, N. O. Combretaceæ; Bhairah, Bahera (*Hind.* and *Beng.*), Behada, Hela (*Bomb.*), Taurik-kay (*Tam.*).—Two cases of accidental poisoning by the fruit of this tree (Belleric myrobalans) have been reported. In one,[2] three boys were poisoned; all, however, recovered. In the other,[3] a woman and two children, one of whom was a weakly girl of eight or nine, were poisoned, and of these the girl died, the two others recovering. The symptoms present were nausea and vomiting, followed by narcotism. The narcotic properties of the fruit reside in the kernel, but it is not known to what they are due.

Identification.—The fresh drupe is obovate, somewhat five angled, about the size of a nutmeg, fleshy, and covered with a grey silky down. When dry it is of the size of a gall-nut, and of a dirty brown colour and astringent taste. It contains abundance of tannin. The stone is hard, and encloses a sweet oily kernel.

Trianthema monogyna, N. O. Ficoideæ; Khapra (*Bomb.*), Nasurjanghi (*Duk.*), Sabuni (*Beng.*), Sharunnay (*Tam.*), Ghelijehroo (*Tel.*).—The root of this, a common weed on rice fields, is used in native medicine as a cathartic. Edgeworth, quoted by Chevers, states that the stem of Trianthema pentandra, closely allied to T. monogyna, is given internally and used locally as an abortifacient, by the village midwives about Jullundur, and that it is known under the names of Bis-kopra and Itsit.

Common English plants mentioned by various writers as irritant or narcotico-irritant poisons, and which may be included in this sub-class, are—Ilex aquifolium or holly, N. O. Aquifoliaceæ; Sambucus nigra, or elder, and Viburnum opulus or guelder rose, N. O. Caprifoliaceæ; Caltha palustris or marsh marigold, N. O. Compositæ; and Ligustrum vulgare or privet, N. O. Oleaceæ. To this sub-class also belongs Spigelia root, the root of Spigelia Marilandica, Maryland or Indian pink root, N. O. Loganiaceæ, a native of the S. United States. This is an irritant cathartic, and in overdoses a narcotico-irritant poison. In this place also may be mentioned cotton root bark, the bark of the root of Gossypium herbaceum, and other species of gossypium. This, officinal in the U.S.P., is said to cause contraction of the uterus and to be a substitute for ergot.

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[1] Blyth on Poisons, p. 422. [2] Chevers, Med. Jurisp., p. 273.
[3] Bo. Chem. Analyser's Rep., 1878–79, p. 14.

Appendix to Vegetable Irritants.

Abrus precatorius, N. O. Leguminosæ; Indian liquorice plant or Jequirity, *vern.* Gunj, Gungchi (*Hind.*), Gunza (*Bomb.*), Gundumani (*Tam.*), Charroti (*Guz.*).—The root of this plant is officinal in the Pharmacopœia of India as a substitute for liquorice, and the seeds are used as small weights by jewellers, a "*rati*" being the weight of one seed. The seeds, roots, leaves, &c., of this plant may be swallowed in considerable quantity without any ill effect being produced. A small quantity, however, of a decoction of the seeds or of other portions of the plant, injected under the skin of an animal, rapidly proves fatal, producing general depression, drowsiness, fall of temperature, and hæmorrhagic lesions. Warden, of Calcutta, found that half a grain of the seed subcutaneously injected, was sufficient to kill cats in twenty to forty hours, and states that one and a half to two grains is sufficient to kill cattle within forty-eight hours. A cold infusion of the seeds applied to the conjunctiva causes purulent inflammation. Warden attributes the activity of the seeds to a proteid substance which he has named abrin. In some parts of India, small sharp-pointed spikes—suis or sutaris—made of a paste prepared by pounding the decorticated seeds with water, and hardened by exposure to the sun, are used for the purpose of destroying cattle. When so used, two of the suis, which are about three-quarters of an inch in length, are inserted into holes in a wooden handle. A blow is then struck with great force, driving the sui protruding from the handle into the animal's flesh, where it is left, and soon causes death. In animals killed by abrus poisoning, œdema of the subcutaneous tissue is found at the seat of the injury if the animal has lived over twenty-four to thirty hours. The mucous membrane of the stomach and intestine is found highly injected, and numerous hæmorrhagic points are seen on the surface of the mucous membrane of the intestine, and also in the interior of vascular organs, *e.g.*, the lungs, liver and spleen.[1]

Identification.—The seeds are spherical, about the size of a pea, polished, and of a bright scarlet colour with a large black spot at one end, other varieties are white or blackish. Their average weight is about one and three-quarters to a little over two grains.

[1] Warden and Waddell on the Non-bacillar Nature of Abrus-Poison. Calc. 1884.

CHAPTER XVII.

IRRITANT POISONS (*concluded*). ANIMAL AND MECHANICAL IRRITANTS.

CLASS V.—ANIMAL IRRITANTS.

Cantharides.

CANTHARIDES, the dried **Cantharis vesicatoria**, blister beetle, or Spanish fly, applied to the skin causes irritation and vesication, and when swallowed or absorbed into the system in poisonous doses, gives rise to the usual symptoms of irritant poisoning, accompanied, when the poison has been taken by the mouth, by blistering of the mouth and throat. The evacuations usually contain blood. Special symptoms in poisoning by cantharides are: (*a*) salivation with swelling of the salivary glands and back of the throat, rendering swallowing painful and difficult, and (*b*) strangury, hæmaturia, inflammation of the genitals, and other similar symptoms, due to the special irritant action of the poison on the kidneys and urinary passages. In fatal cases convulsions generally precede death; insensibility may or may not be present. Cantharides is seldom administered with homicidal intent; cases of poisoning by it are usually accidental, or arise from its being given as an abortifacient, or as an aphrodisiac. As an abortifacient, cantharides often fails, but sometimes succeeds owing to the violent constitutional disturbance produced. There is no proof that the drug has any specific action on the uterus. As regards aphrodisiac action, the drug may possibly excite sexual passion, but it is only likely to do so when given in quantity sufficient to endanger life, or cause serious symptoms. Taylor[1] mentions an English case (*R.* v. *Wilkins*, Liverpool Lent Assizes, 1861) where a man was tried and convicted of administering powdered cantharides to a woman, in which the question arose whether or no an offence had been committed, seeing that the drug had been given solely with the motive of exciting sexual desire. Mayne, in his commentary on sect. 328 of the Indian Penal Code (see *ante*, p. 111), refers to this case as follows:—"In a case under a similar English statute, where it appeared that the prisoner had administered a drug to a female

[1] On Poisons, p. 529.

with intent to excite her sexual passions, in order that he might have connection with her, the conviction was affirmed."

Dose, &c.—The medicinal dose of powdered cantharides is one to two grains, and of the tincture (strength one to eighty) five to twenty minims. The smallest fatal doses recorded are of the powder, twenty-four grains; and of the tincture, one fluid ounce. Recovery has, however, taken place from six ounces of the tincture, and in another case from sixty grains of the powder. The shortest fatal period recorded is twenty-four hours, and the longest (from one ounce of the tincture) seventeen days. Taylor mentions a case where death, with the usual symptoms of cantharides poisoning, resulted in five days from the external application of cantharides ointment in mistake for sulphur ointment, as a cure for itch. **Treatment.**—General. Oily demulcents should be avoided, as cantharidin, the active principle, is soluble in oil. Opiate injections into the bladder, opium suppositories, and warm baths should be used to relieve pain. **Post-mortem appearances.**—These are usually, signs of inflammation of the alimentary canal, congestion of the kidneys, and inflammation of the urinary passages. When the powder has been swallowed, glittering particles of it may be found adhering to the intestinal mucous membrane. Similar particles may be found in the vomited matters.

Detection.—The powder is greyish-brown in colour, and contains shining green metallic-looking particles. These resist putrefaction, and may be detected in the contents of the intestines after long periods of interment. The active principle, cantharidin, present in the powder to the extent of about one-half per cent., may be extracted therefrom, or from organic mixtures containing it, by taking advantage of the fact that it is soluble in alkaline liquids, but can be removed from these by acidulating them, and shaking them with chloroform. On separating and evaporating the chloroform, cantharidin may be recognised in the residue by digesting this with a few drops of oil, and applying the liquid so obtained to the skin, when, if cantharidin is present, vesication will be produced.

Cantharidin is also present in the **Mylabris cichorii**, or **Telini fly**, officinal in the I.P., as a substitute for cantharides, and possessing exactly the same action. The powder of these differs, however, from cantharides powder in containing no shining particles. Cantharidin has also been found in various other Indian blistering flies, *e.g*, in the Mylabris pustulata and M. punctum.[1]

[1] Many other blistering flies are found in India (see 'Pharmacopœia of India,' p. 277).

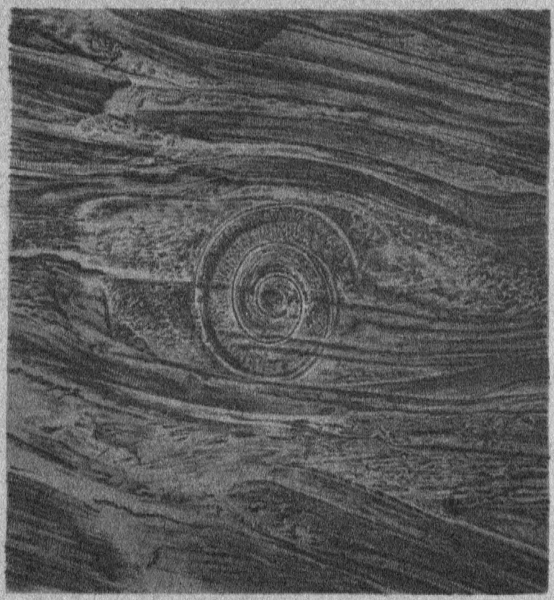
Poisonous Animal Food.

Poisonous Fish.—Many cases, a few of them ending fatally, have been reported, in which persons have been attacked with symptoms of poisoning after eating fish. In some of these cases the symptoms have appeared within a few minutes, in others not for twelve or twenty-four hours, or more, after eating the fish. One or both of two sets of **symptoms** may be present, viz. (1) the usual symptoms of irritant-poisoning, and (2) swelling and inflammation of the eyelids, with profuse lachrymation, accompanied by irritation of the skin and appearance of an eruption resembling nettle-rash. In some cases muscular debility, numbness of the limbs, delirium, and coma have been observed. Death has occurred within the hour, and has been delayed until the ninth day. Apparently, cases of fish-poisoning are divisible into three classes: (1) Exceptional cases attributable to **idiosyncrasy,** the fish eaten proving poisonous only to the individual attacked and not to others. (2) Cases arising from fish **usually non-poisonous,** apparently due to the assumption by the fish of a condition in which it acts as a poison to all. Various theories have been put forward to account for the assumption of this condition. Thus it has been attributed (*a*) to the presence in the fish of copper, derived from the copper sheathing of vessels, &c.; (*b*) to the fish being in spawn; (*c*) to the poisonous nature of food (*e.g.*, acrid mollusca, or acrid spawn), eaten by the fish; (*d*) to the development of a poison by decay (see Ptomaines, p. 308), &c., &c. Cases of these first two classes appear to be most frequently due to shell-fish, especially mussels and oysters, but have arisen from other shell-fish, and from herrings, eels, mackerel, &c. (3) Cases arising from eating fish, certain parts of which seem to be, especially in hot climates, nearly **always poisonous.** To this class belong: (*a*) A case cited by Blyth which occurred at the Cape of Good Hope, in which two adults died within twenty minutes from eating the liver of the toad or ball bladder (diodon)[1]: and (*b*) a case reported by Dr. Collas, of Pondicherry,[2] where three persons were poisoned by eating the Gobus criniger. With reference to this last case Dr. Collas remarks that, although these fish are usually eaten, the native females take great care, in preparing them, to remove the head and intestines, and wash the fish thoroughly.

It may also be mentioned that the secretion of the skin glands of certain species of amphibia has been found to be poisonous. Blyth mentions that a poisonous alkaloid has been found in the skin secretion of the Salamandra maculosa, also in that of the Triton cristatus, or water salamander, and in that of the common toad.

[1] On Poisons, p. 447. [2] Chevers, p. 299.

XXVIII.



Page 227. TRICHINA SPIRALIS IN HUMAN MUSCLE × 130.

From Micro-Photograph by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

Poisonous Meat.—Symptoms more or less resembling those of poisoning, may arise from eating the flesh of diseased animals, and also from eating meat which has undergone decomposition. In cases of the first description, the disease may or may not consist in the presence of a parasite in the meat. The parasites, the presence of which in meat most commonly gives rise to cases, are the trichina spiralis and the various cysticerci.

Trichina spiralis.—Meat affected by this parasite contains, lying among the muscular fibres, small oval sacs, just visible to the naked eye,[1] each containing a coiled-up trichina (see Fig. 28). Sometimes these sacs are so numerous as to give the meat a white speckled appearance. When meat thus affected is eaten without having been thoroughly cooked, a train of symptoms known as trichinosis is produced. The pathology of trichinosis is briefly as follows:—The trichina while enclosed in its cyst is in a chrysalis condition. When the cysts are taken into the stomach or intestines of a warm-blooded animal, the trichina leaves the cyst and begins to produce young. Six to eight days after the ingestion of the cysts, these young trichinæ begin to leave the parent animal and to migrate through the wall of the intestines into the muscles, where they in their turn become encysted, and live by preying on the muscular fibre. The symptoms of trichinosis resemble to a certain extent those of irritant poisoning, differing from an ordinary irritant case chiefly as follows:—(1) There is generally a considerable interval, corresponding to the period of incubation of the worm, between eating the food and full development of the symptoms. (2) Although the pain in trichinosis is intense, the vomiting and purging are not very severe; and the pain is not confined to the stomach and intestines, but extends to the muscles as well, owing to the invasion of these by the trichinæ. (3) In trichinosis, pneumonia is almost a constant symptom; there is often, also, peritonitis, and sometimes paralysis of the muscles. Several outbreaks of trichinosis have been reported, chiefly in Germany, the most notable being one which occurred in 1863, in which 103 persons were attacked, of whom eighty died. There is no remedy, but it should be noted that trichinæ are destroyed by exposure for some time to the temperature of boiling water, and hence thorough cooking of trichinous meat prevents the occurrence of trichinosis.

Trichinæ are more frequently found in the flesh of the pig than in that of any other animal, hence cases of trichinosis have generally occurred from eating imperfectly-cooked pig's flesh in some form or other. In affected animals trichinæ, it is said, will always be found in the muscles of the eye.

[1] Taylor gives the measurement of the sacs as $\frac{1}{50}$th of an inch long by $\frac{1}{100}$th of an inch broad. The worm varies in length from $\frac{1}{50}$th to $\frac{1}{8}$th of an inch. As many as 6000 may be found in about sixteen grains of the affected meat.

Cysticerci.—These are the larvæ of tapeworms, and may be found in the flesh of all animals. Flesh containing them is popularly termed measly, *e.g.*, measly pork. In affected meat the cysticerci are seen as little sacs, filled with fluid, embodied in the muscles. From one side of the interior of the sac a neck projects, terminating in a head surmounted with a crown of hooks (see Figs. 29 and 30). The pork cysticercus (C. cellulosæ) varies in size from a pea to a large bean, and develops into the Tænia solium. The cysticercus of ox flesh is smaller in size and develops into the Tænia mediocanellata. Both these tapeworms affect the human subject. In rare cases also, the larval form of T. solium has been found in the human body. Another variety of tapeworm, T. echinococcus, is only known to affect the dog and wolf; the larval form of this tapeworm, however, affects other animals; *e.g.*, sheep, in which it specially affects the brain, causing "staggers," and the human subject, giving rise to hydatid tumours. Woodman and Tidy remark that echinococcous disease is alarmingly common in Iceland.

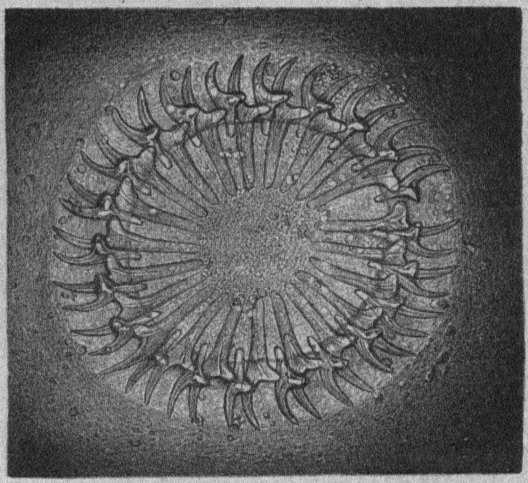
Other diseased Meat.—The flesh of animals affected with disease not specifically parasitic in character, may also, when eaten, give rise to symptoms resembling those of poisoning. Thus, Dr. Letheby met with a case in London, in which sixty-four persons, of whom one died, were attacked with sickness, diarrhœa, and great prostration of strength, after eating sausages made from diseased beef. In another English case, which occurred at Welbeck, in 1880, over seventy persons, of whom four died, were attacked with similar symptoms after eating ham, found, on examination, to contain a bacillus. In the great majority of the Welbeck cases, of which particulars could be obtained, the symptoms did not appear until twelve hours or more after eating the affected food.

Putrid Meat.—Several cases have been reported of persons having been attacked with symptoms of poisoning either irritant or narcotico-irritant in character, after eating putrid meat. In some of these cases the meat eaten has been in a condition of incipient putrefaction only. Cases of this last kind have frequently occurred from eating sausages: hence the term sausage-poisoning.

In sausage-poisoning, besides the usual irritant symptoms, dryness of the skin and mucous membranes, dilatation of the pupils, and paralysis of the upper eyelids have been observed. Various theories have been put forward to account for such cases. Very possibly they are due to the development of a ptomaine or cadaveric alkaloid (see p. 308).

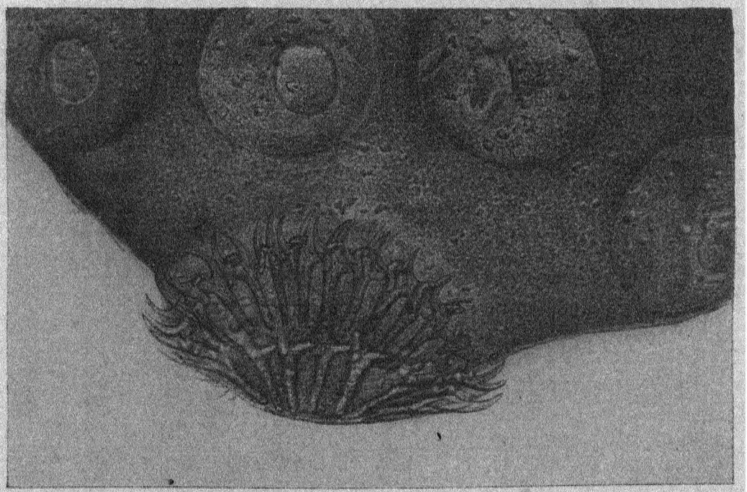
Cheese and Milk.—In more than one case symptoms of irritant poisoning have been produced by eating cheese. In such cases the cheese eaten has generally, but not invariably, been found to be rancid. The milk of diseased cows also has been

XXIX.



HOOKLETS OF CYSTICERCUS × 63.

XXX.



Page 228.

HEAD OF CYSTICERCUS × 63.

From Micro-Photographs by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

known to cause vomiting, and cases have been lately reported of outbreaks of poisoning traceable to milk, the symptoms being irritant in character, and accompanied by burning pain in the œsophagus. In these last referred to cases, and also in a lately reported case of cheese-poisoning, a poisonous ptomaine was found in the milk and cheese at fault.

CLASS VI.—MECHANICAL IRRITANTS.

Under this head may be classed all substances which are liable, when swallowed, to cause symptoms of irritant-poisoning, solely in consequence of their mechanical action on the parts with which they come in contact. Many definitions of the term "a poison" exclude such substances. As already pointed out, however, the question whether or no such substances may properly be called poisons, is for medico-legal purposes in India a matter of little importance. In India, in fact, when it is alleged that an individual has committed an offence, by administering or attempting to administer one of these substances, *e.g.*, pounded glass; the questions which a medical expert has to consider are: (1) What has been the effect of the administration of the substance? and (2) Is the substance one which it is "deleterious to the human body to swallow,"[1] or an "unwholesome thing?"[2] and not, Is the substance "a poison"? Substances which, when swallowed, may act as mechanical irritants, are: (1) Hard, sharp angular or pointed solid matters, *e.g.*, pounded glass, pins, and needles (see also *Arums*, p. 212). (2) Substances which swell largely by imbibition of water, *e.g.*, sponge; and (3) Liquids at a high temperature, *e.g.*, boiling water or melted lead. Of these, the following require special notice:—

Pounded Glass.—This, in many parts of India, is popularly believed to be a very active poison, and has been used both in attempts at suicide and in attempts at homicide. The Bombay Analyser's records for the ten years ending 1884 show that, during that period, this substance only was detected in thirty-one cases of alleged attempted human poisoning. In twenty-three of these it was detected in bread, sweetmeat, or some other article of food; in three more in vomited matters, two of these being cases of attempted suicide by females; in one case it was found after death in the contents of the stomach of a man (*Case* B 31 *e*); in another in some pills; and in the three remaining cases pounded glass, *per se*, was sent for identification. In nearly all these cases, the glass found was coloured glass, resembling fragments of bangles; and in two only it was reported that the individuals suspected of having used the glass with criminal intent were males. Harvey[3] again mentions five cases, all from the Central

[1] Ind. Pen. Code, ss. 324 and 326. [2] *Ibid.*, s. 328.
[3] Beng. Medico-legal Report for the three years ending 1872, p. 292.

Provinces, of alleged attempted homicide by pounded glass, all being alleged attempts by wives to poison their husbands; and Chevers[1] mentions a case brought to the notice of the Chemical Examiner, Bengal, in which a servant attempted to poison his master by pounded glass introduced into a mess of spinach, and also a Bombay case, in which a man seized in the act of committing a robbery, attempted suicide by swallowing fragments of a wine bottle.

The more finely glass is pounded, the more likely are the particles to become completely enveloped in mucus, &c., and to be thus prevented from injuring the mucous membranes. Hence, as the ill-consequences arising from swallowing pounded glass, are solely due to the mechanical injury it inflicts, the more finely it is pounded, the less likely is swallowing it to cause harm. Considerable quantities of pounded glass, in large angular fragments even, have often been swallowed without ill effects being produced. On the other hand, cases are recorded where swallowing pounded glass has caused symptoms of irritant poisoning (see *Cases* B 31 *a* and *b*), and there is reason to suppose that, in exceptional cases, swallowing pounded glass may even cause death (see *Cases* B 31 *c*, *d*, and *e*).

Treatment. This should consist in the administration, first of bulky food so as to envelope the fragments, and then of emetics and laxatives.

Diamond dust.—Diamonds and diamond dust are popularly believed in India to be very poisonous. Thus, in the Baroda case (*Case* B 21), a mixture of arsenious oxide and diamond dust was employed, and Chevers[2] mentions two Indian cases of attempted suicide by swallowing an unbroken diamond. Like pounded glass, any injurious action possessed by diamonds or diamond dust is solely mechanical.

Chopped Hair.—This also may act as a mechanical irritant. Chevers[3] mentions that a belief exists in some parts of India that "tiger's smellers" are poisonous, and states, on the authority of Baboo Kanny Loll Dey, that chopped hair is sometimes used by cattle poisoners.

Boiling Water.—Several English cases of accidental death of children from swallowing boiling water have been recorded, the fatal result being due to suffocation arising from injury and inflammation of the larynx.

Melted Lead.—Swallowing melted lead has caused death. Christison mentions a case which occurred at the burning of the Eddystone lighthouse. A man looking up at the fire with his mouth open, swallowed a quantity of melted lead which fell from the building, and died in twelve days. Seven and a half ounces of lead had reached the stomach, which was severely burnt and ulcerated.

[1] Med. Jurisp., p. 287. [2] *Ibid.*, p. 289. [3] *Ibid.*, p. 291.

CHAPTER XVIII.

CEREBRAL POISONS—CYANOGEN AND BENZENE GROUPS.

CEREBRAL POISONS.

Poisons of this order act mainly on the brain, causing one or other or both of two sets of symptoms. These, in order of their occurrence, are: (1) Excitement with quick pulse and flushing of the face, often accompanied by delirium, and more or less derangement of voluntary movement; and (2) Sopor, deepening into more or less profound coma, with laboured and stertorous breathing. In some poisons of this order, viz. the **mydriatic deliriants**, the production of the first of these two sets of symptoms, accompanied by dilatation of the pupils, is the prominent effect. The majority of poisons of this order—**narcotic poisons**—produce the second set of symptoms as their prominent effect. In poisoning by some of these, *e.g.*, alcohol, a first stage of excitement and delirium usually precedes the second or comatose stage; but when the dose is large, this first stage may be absent. In others, *e.g.*, opium, the first stage is commonly absent, or only slightly marked; but, if the dose is small, or the patient habituated to the action of the poison, a well-marked first stage may be present. **A** few, *e.g.*, hydrocyanic acid and the poisonous cyanides, **act with extreme rapidity**, paralysing the heart if the dose is large, and causing death by syncope; or in smaller doses, acting on the spinal cord as well as the brain, and although producing insensibility, quickly causing death by asphyxia due to paralysis of the muscles of respiration, and not by coma as is usual in cerebral poisoning.

For the great majority of cerebral poisons no chemical antidote is available. Hence, in most cases, the **treatment** must consist in the use of measures directed to (*a*) procuring elimination of the poison, and (*b*) counteracting its effects. If the poison, as is usually the case, has been administered by the mouth, the first of these two indications may be carried out by giving emetics, or using the stomach-pump; the latter being resorted to in preference to repetition of the emetic, seeing that many of these, *e.g.* zinc sulphate, if not ejected by vomiting, are liable to become absorbed and act as poisons. The measures by which

the second indication of treatment may be carried out vary according to the poison and the prominent symptoms. Thus, cold affusions, keeping the patient roused, sinapisms, &c., are indicated when narcotism has to be overcome; and galvanism and artificial respiration when death threatens to occur by asphyxia. In the case of some cerebral poisons, special physiological antidotes are indicated, *e.g.*, atropia in opium poisoning.

For descriptive purposes, cerebral poisons may conveniently be arranged in six groups, viz.: 1. Cyanogen group; 2. Benzene group; 3. Alcoholic group; 4. Narcotic gases; 5. Vegetable narcotics; and 6. Mydriatic deliriants.

GROUP 1.—CYANOGEN AND ITS DERIVATIVES.

Hydrocyanic or Prussic Acid, HCN or HCy.

Poisoning by hydrocyanic acid, common in England, was some years ago almost unknown in India. Of late years, however, a few suicidal cases have been reported. Pure undiluted hydrocyanic acid rapidly decomposes, and is not met with in commerce. Dilute hydrocyanic acid, the form in which the acid is used as a poison, is officinal in the Pharmacopœias. The dilute acid of the British and Indian Pharmacopœias contains 2 per cent. of anhydrous hydrocyanic acid; that of various foreign Pharmacopœias ranges from this strength to 10½ per cent. (Paris Pharmacopœia), or even more. An acid met with in commerce called Scheele's acid, contains 5 per cent. Certain portions of many plants contain hydrocyanic acid, or yield it under appropriate treatment, owing to the decomposition of amygdalin, or substances allied to amygdalin (see *post, Essential oil of bitter almonds*). Hydrocyanic acid swallowed, inhaled in the form of vapour, or otherwise introduced into the system, paralyses both the brain and spinal cord, causing insensibility and loss of muscular power. Death, from large doses, occurs rapidly by syncope, due to arrest of the heart's action; or from smaller doses, less rapidly by asphyxia, due to paralysis of respiration.

Symptoms.—These vary to a certain extent with the dose. **Small poisonous doses** cause a hot bitter taste, giddiness, pains in the head, and confusion of intellect, followed by insensibility and loss of muscular power. The eyes are bright and prominent, the face pale, and salivation with ulceration of the mouth is frequently present. The breathing often becomes stertorous, the breath smells of hydrocyanic acid, and in a very short time (see *fatal period*) death takes place by asphyxia. Lock-jaw and tetanic convulsions, and involuntary expulsion of urine and fæces, often precede death. Vomiting has been observed, but is not a common symptom. **Large poisonous doses** cause almost immediate insensibility and rapid death from syncope. In rapidly

fatal cases convulsions are not usually present, but there may be involuntary expulsion of urine and fæces. In cases of this class, death usually takes place with a forcible expiration, which may or may not be accompanied by a shriek. Some of the more important medico-legal questions which may arise in case of poisoning by hydrocyanic acid, are as follows:—

1. **What interval may elapse between swallowing the poison and supervention of insensibility.**—Large doses given to animals cause almost immediate insensibility. In man, the action of the poison appears to be less rapid; insensibility may, however, come on in a few seconds, and is rarely, if a full dose has been taken, delayed beyond the second minute. Still, however, even when a full dose has been swallowed, considerable power of volition and locomotion may remain, and various acts may be performed in the short interval between swallowing the poison and supervention of insensibility. Hence, finding the bottle, out of which the poison has been taken, corked, or even placed on a shelf or table close to but out of reach of the body, is consistent with a supposition of suicide.

2. **As to the shriek.**—This is not nearly so frequent a symptom of hydrocyanic acid poisoning in man, as it is in the lower animals. What may be called the true hydrocyanic acid shriek accompanies the last forcible expiration; after such a shriek power of speech, &c., no longer remains.

3. **Presence of hydrocyanic acid in various articles of food.**—Amygdalin, yielding hydrocyanic acid by its decomposition, is contained in the seeds, leaves, and flowers, and sometimes the bark, of most species of the sub-orders amygdalæ and pomeæ of the N. O. Rosaceæ. Its presence in the following may be specially noted: in bitter (but not in sweet) almonds, in apple and pear pips; in plum, damson, cherry, peach, apricot, and quince kernels; and also (apparently) in loquat seeds. The presence of hydrocyanic acid ready formed, in the root of the jatropha manihot, has already been mentioned. It is estimated that 240 grains of bitter almond pulp, 333 grains of cherry kernels, and from about 1200 to 2200 grains of apple pips, are required to yield a quantity of hydrocyanic acid equal to 30 minims of the B.P. dilute acid.[1] Kirschwasser, a brandy distilled from wild cherries, contains hydrocyanic acid to the extent, it is said, of 1 to 4 grains in a pint. Hydrocyanic acid is also contained in chlorodyne (see p. 259).

Fatal period and dose.—Large doses have been found to kill the lower animals almost instantaneously. In man death occurs less rapidly, but has occurred as early as the second minute, and as late as one and a half hours after swallowing the poison. When the dose is 1½ drachms or more of the B.P. acid,

[1] Peach kernels contain rather less amygdalin than cherry kernels; plum kernels contain rather more amygdalin than apple pips.

the average fatal period is two to ten minutes. The smallest dose which has proved fatal to an adult is 0·9 grains of anhydrous acid, death occurring in twenty minutes; recovery has, however, taken place from 2·4 grains. One grain of the anhydrous acid may, but will not necessarily, prove fatal. In estimating the amount taken, it is important to recollect that drops and minims are not necessarily the same. Woodman and Tidy state that 10 drops of hydrocyanic acid equal on an average 20 minims.[1] It may be further noted that dilution seems to make no difference to the action of the poison, but exhaustion from any cause, such as fatigue, favours its action; also that, although it has been asserted that hydrocyanic acid may act as a cumulative poison, the weight of evidence is greatly against its so acting.

Treatment.—The best antidote is a mixture of a ferrous and ferric salt, with a little caustic soda or potash, or, if caustic alkali is not obtainable, with carbonate of soda. Inhalation of chlorine, *e.g.*, from a mixture of chloride of lime and water held near the nostrils, is also useful. The other indications are to promote vomiting, to endeavour to restore sensibility by cold affusion and inhalation of weak ammonia, and to employ artificial respiration.

Post-mortem appearances.—These may be nil, but are generally similar to those of death from asphyxia. The odour of hydrocyanic is often, but not always, perceptible in the body, in the brain and muscles, as well as in the stomach. The smell of hydrocyanic acid has been detected in the stomach seven or eight days after death.

Detection.—Hydrocyanic acid gives (1) with silver nitrate solution a white crystalline precipitate soluble in hot strong, but not in dilute nitric acid. The dried precipitate is decomposed by heat with evolution of an inflammable gas (cyanogen), metallic silver being left. (2) On adding caustic potash solution, followed by a mixture of a ferrous and ferric salt, and then by dilute hydrochloric acid in quantity sufficient to dissolve the precipitated oxides of iron, a blue precipitate (Prussian blue) will subside. (3) If mixed with a little yellow ammonium sulphide, and the mixture evaporated to dryness, the residue will be found to give a blood-red colour with ferric chloride solution, discharged by solution of mercuric chloride, but not by dilute hydrochloric acid. **In organic mixtures** hydrocyanic acid may be detected by distillation on a salt-water bath, and testing the distillate as above. Or the matters may be warmed and the tests applied to the vapour, receiving this in watch-glasses containing a drop of fluid, and inverted over the mouth of the vessel containing the matters under examination. The fluid in which the vapour is received should be silver nitrate solution for test (1), caustic potash

[1] According to the same authorities, ten drops of chloroform or of tincture of opium equal five to six minims, and ten drops of the following tinctures equal six to eight minims:—aconite, digitalis, and hyoscyamus.

solution for test (2), and yellow ammonium sulphide for test (3). If the fluid is alkaline, it must be first acidulated with dilute sulphuric acid, so as to liberate the hydrocyanic acid. The quantity present may be estimated in the distillate by precipitation as cyanide of silver; long-continued distillation may be required in order to separate the whole of the poison.

If the result of the analysis is non-detection, death may nevertheless have been due to poisoning by hydrocyanic acid. A case is recorded of death from hydrocyanic poisoning, in which analysis twenty-six hours after death failed to detect the poison. On the other hand, it has been detected by analysis seventeen, twenty-one, and even twenty-three days after death, and may be detected even if no odour of the acid is perceptible.

If the result of the analysis is detection, it may possibly be objected that the poison found (*a*) has been yielded by apple pips, cherry kernels, or the like, hence the contents of the stomach, vomited matters, &c., should always be carefully searched for such bodies, which, if found, should be separated before proceeding with the analysis; (*b*) has been yielded by the decomposition of sulphocyanide of potassium present in the saliva; this theory may account for the discovery of a minute trace of hydrocyanic acid, but not for more; (*c*) has been formed as the result of putrefaction; hydrocyanic acid, however, has never been known to be formed in this way; or (*d*) has been produced by the action on organic matters of the heat employed in distillation. A high temperature, much higher than that of a salt-water bath, would be required, however, to produce even traces in this way.

Essential oil of bitter almonds, Benzyl hydride, or Benzoic aldehyde, C_7H_6O, is obtained by distillation of an emulsion of the cake left after expression of the fixed oil from bitter almonds. It is formed by the fermentative action of emulsin, present both in sweet and bitter almonds, on amygdalin, a glucoside present in the bitter, but not in the sweet variety.[1] During the decomposition hydrocyanic acid is also produced, which, if not removed, renders the oil poisonous. Essential oil of bitter almonds unpurified, as generally sold, contains 8 to 15 per cent. of hydrocyanic acid. Seventeen drops of the unpurified oil has caused death in an adult, and probably less would prove fatal. Essential oil of bitter almonds is also sold under the name of peach-nut oil, and a fatal case is reported, arising from its having been sold by mistake for beech-nut oil. Diluted with four to eight parts of rectified spirit, it forms the almond flavour or essence of the shops, sold for the purpose of flavouring confectionery. Bitter almond water,

[1] The subjoined formula shows the decomposition:—

$$\underset{\text{Amygdalin}}{C_{20}H_{27}NO_{11}} + 2H_2O = C_7H_6O + HCN + \underset{\text{Glucose.}}{2C_6H_{12}O_6}.$$

another preparation, contains hydrocyanic acid to the extent of 0·25 to 1·0 per cent. A fatal case of poisoning by **bitter almonds** in a female, æt. 31, is also reported. The quantity taken was estimated at about 1200 grains. The symptoms, treatment, &c., in poisoning by essential oil of bitter almonds, are the same as in poisoning by hydrocyanic acid. Hydrocyanic acid may be detected in it by the vapour tests, or by applying the tests for the acid, to water which has been shaken with the oil. The purified oil—from experiments on animals—acts as an intoxicant, but is very much less poisonous than the crude oil.

Waters distilled from the following also contain hydrocyanic acid, derived from decomposition of amygdalin, or a substance allied to it:—the leaves of the **cherry laurel** (Prunus laurocerasus); the flowers, bark, seeds, and leaves of the **mountain ash** (Sorbus aucuparia); and the bark, seeds, and leaves of the **cluster cherry** (Prunus padus). One ounce of cherry laurel water has proved fatal to an adult, and in a celebrated case (murder of Sir T. Broughton, 1781) two ounces proved fatal in half an hour. The **blossoms of the peach** also have, from a similar cause, in two cases caused death.

Cyanogen and certain derivatives of Cyanogen.

Cyanogen Gas, CN or Cy, has been found by experiments on animals to be intensely poisonous, causing convulsions, dilated pupils, coma, and death in five to six minutes. So also are liquid **Cyanogen chloride**, CNCl, solid cyanogen chloride, $C_3N_3Cl_3$, and **methyl cyanide**, CH_3CN. No cases of poisoning by these substances in man have been reported.

The Cyanides of Potassium, Sodium, and Ammonium are all intensely poisonous. Cyanide of potassium, KCN, more commonly met with than the others, contains cyanogen equal to about 40 per cent. of hydrocyanic acid. It is largely used for various purposes in the arts, *e.g.*, in cleaning gold and silver lace, plate, &c.; by photographers for removing silver stains; and by electro-platers, the ordinary electro-plating solution being silver cyanide dissolved in cyanide of potassium solution. Fatal cases have been reported from swallowing this solution as well as from swallowing cyanide of potassium; and serious symptoms have arisen from the absorption, through abrasions on the skin, of cyanide of potassium, employed for the purpose of removing silver stains from the hands. 2½ grains of pure potassium cyanide may be regarded as a minimum fatal dose. The commercial salt is, however, generally impure from the presence of potassium carbonate, produced by the action of the carbon dioxide of the air on the cyanide. In an exceptional case recovery took place after swallowing more than half an ounce of the commercial salt. The symptoms, &c., are the same as in

poisoning by hydrocyanic acid. Probably, however, after death more evidence of irritation will be found.

Mercuric Cyanide.—This, already mentioned as a poisonous mercuric salt, according to some authorities, acts like hydrocyanic acid. **Silver Cyanide** also, from experiments on animals, appears to act like hydrocyanic acid, but is much weaker; it contains cyanogen equal to about $\frac{1}{4}$th of its weight of hydrocyanic acid. A case of attempted suicide by swallowing cyanide of silver, in which recovery took place under prompt treatment, recently occurred near Poona.

Potassium Ferrocyanide.—Yellow prussiate of potash, K_4FeCy_6, under ordinary circumstances is either not poisonous, or only very feebly poisonous. When acted on by strong acids, however, it yields HCy. In one case, death resulted from swallowing a dose of this salt followed by one of tartaric acid; and in another, from swallowing a dose of the salt, followed by a mixture of nitric and hydrochloric acids. Other ferrocyanides probably act similarly to potassium ferrocyanide.

Potassium Sulphocyanide is poisonous, but not very active. The **Cyanates** (from cyanic acid, HCNO) are asserted to be non-poisonous.[1] **Cyanuric acid,** however, $H_3C_3N_3O_3$, Blyth states, causes symptoms and effects similar to those produced by hydrocyanic acid.

GROUP 2.—BENZENE GROUP.

Benzene, C_6H_6, or Benzol, a liquid prepared by distillation from coal tar naphtha, used in the arts as a solvent, is a powerful narcotic poison. Inhalation of its vapour has caused narcotic effects with twitchings of the muscles and convulsions. Taylor[2] records a case of a boy who swallowed about three ounces of coal naphtha and died in three hours. The symptoms were delirium, followed by coma with contracted pupils. There was complete loss of muscular power and great difficulty of breathing.

Nitro-Benzene, artificial oil of bitter almonds, or essence of mirbane, $C_6H_5NO_2$, obtained by the action of strong nitric acid on benzene, is a liquid possessing the same odour as hydrocyanic acid, and a powerful narcotic poison. The symptoms produced by it are exactly the same as in poisoning by hydrocyanic acid, but with one remarkable point of difference, viz.: that there is an interval generally of at least two hours, but sometimes longer, between swallowing or inhaling the poison, and first appearance of the symptoms. Several deaths have been reported from swallowing nitro-benzene. In one case quoted by Taylor,[3] a boy, æt. thirteen, swallowed a small quantity: no symptoms appeared for several (apparently eight) hours; he then

[1] Hünefeld, quoted by Woodman and Tidy, For. Med. p. 474.
[2] On Poisons, p. 656. [3] *Ibid.*, p. 666.

suddenly became insensible, and died four hours after the attack, or twelve hours after swallowing the poison. Blyth considers it probable from recorded cases, that 15½ grains, or even less, would, if swallowed, prove fatal to an adult. Death also has occurred from inhalation of the vapour as in a case reported by Dr. Letheby,[1] in which a man, æt. forty-three, having accidentally spilt some nitro-benzene over his clothes, became comatose in four and died in nine hours. Bad effects even are said to have arisen from washing with soap scented with nitro-benzene, especially when hot water has been used.

Aniline or Phenylamine, $NH_2 (C_6 H_5)$, obtained by the action of nascent hydrogen (which may be evolved by the action of acetic acid on iron) on nitro-benzene, is a powerful narcotic poison, acting similarly to hydrocyanic acid. It is remarkable that the sulphate of aniline appears to be almost inert to man. Symptoms of poisoning, however, have followed from external application of the hydrochlorate of aniline.

Detection of the above.—Aniline (1) with sulphuric acid and manganese dioxide, or lead peroxide, gives a green colour changing to a persistent blue, and then to black; and (2), with solutions of hypochlorites, *e.g.*, chloride of lime, gives a blue or violet colour, soon changing to brown. **Nitro-benzene** may be recognised by converting it into aniline and applying the above tests. The conversion may be effected by dissolving the nitro-benzene in alcohol, and adding hydrochloric acid and zinc. From the acid liquid, after driving off the alcohol, aniline may be separated by neutralizing the liquid with carbonate of soda, and shaking the neutralized liquid with ether. The ether is then separated, allowed to evaporate, and the residue tested for aniline. **Benzene** is detected by converting it first into nitro-benzene, by treatment with nitric acid, and then into aniline as above. **From organic mixtures** benzene and nitro-benzene may be separated by distillation. If the matters to be distilled are first acidulated with sulphuric acid, aniline, if present, will be found in the residue left in the retort, and may be separated from it by Stas' process (see page 184), using ether as a solvent. Nitro-benzene may become changed in the body into aniline.

Aniline dyes.—These, of various colours, are mostly prepared from rosaniline, a red colouring matter usually manufactured by the action of arsenic acid on aniline, and liable therefore to contain arsenic. There is some reason to believe that certain of these dyes, even if free from arsenic, may produce ill effects. Aniline dyes therefore should never be used for colouring articles of food. They have however been used to colour confectionery, and Chevers mentions the use by native females of red aniline dyes for painting the lips, and refers to a case of poisoning in a child from this practice. Essence of peppermint has been found coloured

[1] Taylor on Poisons, p. 666.

green with an aniline colour. The colour of rosaniline is discharged by ammonia, and restored again by acetic acid.

Carbolic acid, phenic acid, phenol, or phenyl alcohol, C_6H_5HO, obtained from coal tar, is largely used as a disinfectant. It is the active ingredient of many disinfecting powders, *e.g.*, Calvert's, which contains free carbolic acid mechanically mixed with siliceous matter, and separable from it by distillation; and Macdougall's, which contains carbolic acid in combination with lime, calcium sulphite being also present. From Macdougall's powder, carbolic acid may be separated by decomposition with dilute hydrochloric acid. Carbolic acid is a powerful poison, causing when swallowed burning pain in the mouth and throat, whitening and hardening of the lining membrane of the mouth, and occasionally vomiting. Insensibility speedily follows, passing into coma, with stertorous breathing and contracted pupils. The urine is suppressed or scanty, and dark coloured. Death has occurred in ten minutes; the usual fatal period however is one to four hours. One and a half teaspoonfuls of the concentrated acid has caused death, and in four cases out of five, 15 grammes (231½ grains) proved fatal to adults. Dangerous symptoms may be caused by six or seven drops, and death has resulted from its external application. Resorcin, a substance closely allied to carbolic acid, and like it used as an antiseptic, has a similar toxic action.

Detection.—(1) The odour of carbolic acid is characteristic, and may be recognised during life in the patient's breath, urine, or vomit, and after death in the tissues. (2) Slips of some kinds of pine wood moistened first with carbolic acid, and then with hydrochloric acid, acquire a blue colour. The pine wood used should always be tested first: (*a*) with hydrochloric acid only, and (*b*) with carbolic acid and hydrochloric acid, as some varieties give a blue colour with hydrochloric acid only, and others do not give a blue colour with carbolic acid and hydrochloric acid. (3) If to a solution of carbolic acid one fourth of its volume of ammonia be added, and then a minute quantity of a hypochlorite, a blue colour is produced, turning red with acids; warming hastens the development of the blue tint.

The following substances allied to carbolic acid also are poisonous. **Picric or Carbazotic acid** or tri-nitro-phenol, $C_6H_2(NO_2)_3HO$, a yellow crystalline substance, having a bitter taste, and great colorific power; and **Creosote,** a mixture of phenols (aromatic alcohols homologous with carbolic acid) obtained by distillation from wood tar.

With the above may be mentioned the following:—

Petroleum.—This is a mixture, occurring in nature, of hydrocarbons of the paraffin series (homologous with methane, CH_4). From it by fractional distillation are obtained various liquids. Of

these the lighter are known under the names of gasoline, benzoline, mineral naphtha, and petroleum ether or spirit; the intermediate portions form the kerosine oil of commerce used for lighting purposes, and the heavier portions are used for lubricating machinery. A few cases of poisoning by petroleum are recorded, the symptoms being those of narcotic poisoning. Narcotic effects have also been produced by inhalation of the vapour.

Oil or spirits of turpentine, camphine.—Several slightly differing varieties of oil of turpentine exist, all being terpenes, or hydrocarbons of the formula $C_{10}H_{16}$. A few cases of poisoning of children by oil of turpentine, two of them fatal, are recorded by Taylor, the symptoms being those of narcotic poisoning. In the two fatal cases the dose was about half an ounce; the children were respectively three and five months old. Taylor also mentions a non-fatal case of poisoning in an adult by camphine, or rectified oil of turpentine, in which vomiting and purging were present. Turpentine, it may be noted, is excreted by the kidneys and communicates to the urine a smell of violets; painful micturition and hæmaturia may result from its action on the urinary organs.

CHAPTER XIX.

CEREBRAL POISONS—*continued.* ALCOHOLIC GROUP AND NARCOTIC GASES.

GROUP 3.—ALCOHOLIC GROUP.

Alcohol, Ethyl Alcohol, Ethyl hydroxide, C_2H_5HO.

WATER free or absolute alcohol is seldom met with. Rectified spirit, the nearest approach to it in common use, contains 16 per cent. by weight of water. The quantity of real alcohol in liquors sold for potable use is approximately: in strong spirits, such as brandy, whisky, rum, arrack, &c., 37 to 45 per cent.; in strong wines, such as port and sherry, 15 to 22 per cent.; in light wines, *e.g.*, claret and hock, 6 to 9 per cent.; and, in malt liquors, such as porter and ale, 3 to 6½ per cent. Proof spirit (in terms of percentage of which the strength of alcoholic liquors is often stated) contains 49·24 per cent. by weight of absolute alcohol.

In acute alcoholic poisoning, which may arise from the inhalation of alcoholic vapours, as well as from swallowing alcoholic liquids, there is usually but not always a previous stage of excitement and delirium. This is followed by stupor deepening into coma with stertorous breathing. The face is usually flushed, the breath smells of alcohol, and the pupils are generally but not always dilated. As a rule the patient can be temporarily roused into partial sensibility by a loud noise or violent shock. Vomiting and convulsions are occasional symptoms. Occasionally also the symptoms remit, the patient recovers consciousness, but subsequently dies.

The diagnosis of alcoholic poisoning from concussion of the brain, apoplexy, and poisoning by other narcotics, *e.g.*, opium, is sometimes a difficult matter. In these the face is usually pale, and in opium poisoning the pupils are contracted. A smell of alcohol in the breath, it must be recollected, merely indicates the presence of alcohol in the stomach; accompanying narcotic symptoms may, or may not, be due to alcohol poisoning.

Fatal dose, &c.—Blyth estimates, in terms of absolute alcohol, the probable minimum fatal dose as follows:—For a child under twelve, 1 to 2 ounces; for an adult, 2½ to 5 ounces. Recovery has been recorded in an adult after swallowing a quart

of whisky (probably equal to at least 9 ounces of absolute alcohol), and in a child of five after swallowing 3 ounces of rum (probably equal to 1 to 1½ ounces absolute alcohol). In alcohol poisoning, death obviously may occur as an indirect result, as for example from a fall or other accident brought about by intoxication. After coma has supervened, death may occur in a few minutes or not for days. Blyth states that death has occurred after coma of three, four, and even six days' duration.

Treatment.—The contents of the stomach should be at once evacuated, preferably by the stomach-pump; and endeavours should be made to restore sensibility by cold affusion, galvanism, ammonia to the nostrils, &c. Strong coffee may be administered as in opium poisoning.

Post-mortem appearances.—The mucous membrane of the stomach, generally but not always, shows signs of inflammation. These vary from patches of redness, to a condition similar to that found in poisoning by a powerful non-corrosive irritant. The brain is usually congested, so also frequently are the lungs; and the large vessels of the chest are usually found full of dark fluid blood. An odour of alcohol is usually present in the contents of the stomach, and may be noticeable also in other parts of the body.

Chronic alcoholic poisoning.—Long-continued abuse of alcoholic liquors may, as is well known, give rise to disease of various organs, and to delirium tremens. Delirium tremens may be distinguished from intoxication by the history of the case, and the character of the delirium. This may be shortly described as a delirium of dread, accompanied by delusions most commonly connected with visual perception. The patient imagines he sees various objects lying around him, or crawling about, and is often violent, with a tendency to suicide, or less commonly to homicide. As a rule, he can be roused into temporary sanity when sharply spoken to. Legally, delirium tremens is unsoundness of mind,[1] not intoxication.

The criminal responsibility of individuals for acts done while in a state of alcoholic or other intoxication [2] is dealt with in sects. 85 and 86 of the Indian Penal Code. For sect. 85, see p. 2. Sect. 86 is as follows:—"In cases where an act done is not an offence unless done with a particular knowledge or intent, a person who does the act in a state of intoxication shall be liable to be dealt with as if he had the same knowledge as he would have had if he had not been intoxicated, unless the thing which intoxicated him was administered to him without his knowledge or against his will." Mayne, in commenting on this section,

[1] For the criminal responsibility of persons of unsound mind, see Insanity, p. 411.

[2] The responsibility is the same, no matter what the *nature* of the intoxicating agent.

writes: "Sometimes in determining the quality of an offence, evidence is necessary of a specific existing state of mind, which must be found as a fact, and cannot be assumed. For instance, supposing a fatal blow to be struck under circumstances of grievous provocation; it might be shown that, notwithstanding the provocation, the defendant had acted, not under its influence, but from a preconceived malicious resolve to kill. If so, the offence would be murder. But the mere fact of the deadly blow would not be sufficient evidence for that purpose. Given the provocation, the legal inference derivable from the character of the blow would be exhausted in making the act be culpable homicide not amounting to murder. Evidence of a different state of mind would be required to constitute the graver charge. In this state of things, intoxication might be an answer to the charge of murder."

Methyl alcohol, wood spirit, wood naphtha, or pyroxilic spirit, CH_3HO, obtained by the destructive distillation of wood, has an action on the system similar to that of ethyl alcohol. It has a peculiarly disagreeable odour, and is used for fouling rectified spirit, so as to render it non-potable. Rectified spirit, mixed with ten per cent. of pyroxilic spirit, is sold under the name of methylated spirit, for use in arts and manufactures.

Amyl alcohol, fusel oil, or potato spirit, $C_5H_{11}HO$.—This is a higher alcohol of the same homologous series as the two alcohols mentioned above, but, unlike them, is not miscible with water. It is much less volatile than ethyl alcohol, and is found in the last portions of the distillates obtained from fermented potatoes and fermented grain. Like ethyl alcohol, it is a narcotic poison, but is much more powerful in its action, and more prone to cause convulsions. It appears to be more powerfully poisonous when inhaled in the form of vapour, than when swallowed in the liquid form. Carelessly distilled whisky and other spirits are apt to contain amyl alcohol, and, in consequence, to be more powerfully intoxicant and more injurious to health. When such spirits are kept, the amyl alcohol they contain gradually decomposes, fragrant ethers being formed, and the spirit, as a consequence, improves in flavour, and becomes less hurtful.

Detection.—All three alcohols are inflammable, and burn with a pale blue flame. All three, if added to a mixture of bichromate of potash solution and strong sulphuric acid, change the colour of the mixture from red to green. Methyl and ethyl alcohols are both miscible with water. Methyl alcohol has a peculiarly disagreeable odour. Ethyl alcohol has a pleasant odour, and, when heated with sulphuric acid and an acetate, gives off vapours of acetic ether, the odour of which is peculiar and characteristic. Amyl alcohol is not miscible with water; treated as above, it evolves amyl acetate, which has the characteristic odour of jargonelle pears. From organic mixtures, methyl and

ethyl alcohols may be readily separated by distillation. The liquid to be distilled, if acid, should first be neutralized. Methyl alcohol, on account of its superior volatility, should be looked for in the first portions of the distillate. From the distillates the alcohols may be obtained, sufficiently free from water for recognition, by shaking with solid potassic carbonate. The liquid will then separate into two layers, of which the upper one contains the alcohols Amyl alcohol, the boiling-point of which is 132° C., will be found either in the last portions of the distillate, or in the residue in the retort. From the latter it may be removed by shaking with ether, in which it is soluble.

Ether, sulphuric ether, or ethyl oxide $(C_2H_5)_2O$.—This, when swallowed, produces effects similar to those produced by alcohol. It appears to be more rapid and more powerful in its action than alcohol, but its effects pass off more quickly. Blyth estimates that one fluid-ounce swallowed would kill most adults. It is sometimes taken by spirit-drinkers, as a substitute for whisky, brandy, &c. Ether vapour, if inhaled, produces effects similar to those produced by inhalation of chloroform vapour, but is said to be less likely to cause arrest of the heart's action. Inhalation of ether vapour has, however, caused death. A given quantity of ether acts more powerfully, when inhaled in the form of vapour, than when swallowed in the liquid form.

Amyl nitrite, $C_5H_{11}NO_2$.—Inhalation of the vapour of this is employed for the purpose of relaxing vascular spasm. When inhaled, its first effect is to cause flushing of the face, throbbing of the carotids, a quick full pulse, and giddiness. Experiments on animals show that large doses of the vapour cause convulsions, coma, and death.

Chloroform, Methenyl Chloride, $CHCl_3$.

Chloroform, when swallowed, produces effects very similar to those produced by alcohol, causing, if taken in sufficient quantity, coma with stertorous breathing and dilated pupils. As in alcohol poisoning, vomiting and convulsions are occasionally present. Taylor mentions a case in which an adult, who had swallowed three ounces, recovered sensibility in fourteen hours, but died of acute gastritis with collapse, twenty-nine hours after swallowing the poison. The smallest dose of liquid-chloroform which has proved fatal to an adult is 3·8 drachms; a case of recovery, however, is reported after swallowing four ounces. One drachm proved fatal in three hours to a boy æt. four. A given quantity of chloroform acts very much more powerfully when inhaled in the form of vapour, than when swallowed in the liquid condition.

Inhalation of chloroform vapour causes (1) a stage of **excitement**, with flushing of the face and contracted pupils: in this stage, delirium is nearly always present, and sometimes the patient struggles violently. To this succeeds (2) a stage of **complete anæsthesia**, with relaxation of the muscles and suspension of reflex action. This is the stage in which surgical operations are performed; it may be looked on as fully developed, when touching the conjunctiva fails to cause reflex closure of the eyelids. If the inhalation of chloroform is continued, (3) a stage of **paralysis** sets in. Respiration becomes slower and more feeble, the heart's action becomes weaker, and death ensues from paralysis of respiration, or from arrest of the heart's action. Death, however, may occur during any stage of the inhalation, and may be due (*a*) to sudden stoppage of the action of the heart, liable, in exceptional cases, to occur at any stage; or (*b*), to asphyxia, which may be brought about in various ways, *e.g.*, by closure of the glottis, owing to pressure of the tongue, or by blood or vomited matter finding its way into the air-passages. In more than one case, death has occurred within two minutes of the commencement of the inhalation. In one case, thirty drops, and in another fifteen to twenty drops, inhaled in vapour, caused death. A much larger quantity than this (about 3½ drachms) is commonly required to cause anæsthesia. The more concentrated the vapour, the more likely is danger to arise. The death ratio from chloroform during operations is variously estimated at ·75 to 3·4 per 10,000 cases.

Cases occasionally occur in which an individual alleges, that he or she has been rendered insensible by chloroform, and, while in that condition, robbed or raped. As bearing on the question of the truth or falsity of such charges, it may be noted: 1. That chloroform vapour does not cause immediate insensibility, and that it is difficult to administer chloroform to persons against their will, unless considerable force is employed. 2. That to successfully administer chloroform vapour during sleep requires the greatest skill and care, and even then can only be accomplished in a small proportion of cases; and 3. That inhalation of chloroform may cause sexual excitement, accompanied by delusions, remembered after recovery of sensibility, and believed to be real events.[1]

Treatment.—If liquid chloroform has been swallowed, the stomach-pump should be used at once. As regards poisoning by the vapour, it may be first pointed out that chloroform vapour should always be given greatly diluted with air, and on a nearly empty stomach; and that its administration should be avoided in patients suffering from certain cardiac diseases. In cases of poisoning, the patient should be placed in a horizontal position, and cold affusion, artificial respiration, and galvanism employed.

[1] Lauder Brunton's Pharmacology, p. 723.

It should be seen that nothing mechanically impedes respiration, and that the tongue is well forward. It may be necessary to draw it forward with a pair of forceps. Turning the head on one side will often suffice, and will, at the same time, allow of the escape of vomited matters, &c.

Post-mortem appearances.—Not characteristic. The blood is usually dark coloured and very fluid. After death from swallowing liquid chloroform, signs of inflammation of portions of the gastric mucous membrane have been found.

Detection.—Death from chloroform may occur, and analysis fail to detect the presence of poison in the viscera, owing to its having escaped by volatilisation, or to its having become decomposed in the body. This last may be due to the action of alkalies, a formate of the alkali being formed according to the following equation: $CHCl_3 + 4KHO = KCHO_2 + 3KCl + 2H_2O$. From organic mixtures chloroform may be separated by distillation, and recognised in the distillate by its peculiar odour. Or the mixture, first neutralized if acid, may be distilled, and the vapours passed through a glass tube heated to redness. Under these circumstances the chloroform is decomposed with formation of hydrochloric acid and free chlorine. The presence of the first is shown by the vapours reddening moistened blue litmus paper, and producing a white precipitate in silver nitrate solution; and of the second, by the vapours causing a blue colour to appear on paper soaked in a mixture of starch and potassium iodide solutions.

Chloral (Trichlorinated aldehyde), C_2HCl_3O.

This is used in medicine as a hypnotic in the form of chloral hydrate (C_2HCl_3O, H_2O), and has in several cases caused death. It causes deep sleep, followed, if an overdose has been taken, by coma with motor paralysis; and slowing, weakening, and ultimate arrest of the heart's action, and of the movements of respiration. These effects appear to be due to the action of the drug on the nerve-centres, and not on the nerves. The pupils are nearly always contracted. A skin eruption, in some cases resembling that of scarlatina, in others urticaria or purpura, has been observed. Possibly in many cases the fatal result is attributable to the decomposition of chloral within the body into chloroform.[1] This may be effected by the action of alkalies, a formate of the alkali being at the same time formed, thus: $C_2HCl_3O + KHO = KCHO_2 + CHCl_3$. Sometimes a single overdose of chloral causes sudden death by syncope.

Fatal dose.—The probable minimum fatal dose cannot be

[1] From the experiments of Hammersten, it would appear that chloral ordinarily acts without undergoing decomposition into chloroform (see Lauder Brunton's Pharmacology, p. 715).

stated with certainty. In one case thirty grains, or not more than a full medicinal dose, caused the death in thirty-five hours of a woman æt. twenty. Persons, however, have recovered from doses of 150 and 160 grains. The syrup of chloral B.P. contains ten grains in each fluid drachm.

Post-mortem appearances.—Considerable congestion of the vessels of the brain and its membranes has been observed.

Treatment.—Evacuate the contents of the stomach, preferably by the stomach-pump; administer decoction of coffee freely, introducing it by the stomach-pump if necessary; keep the patient warm, and endeavour to restore respiration. Hypodermic injection of strychnia is strongly recommended by several authorities; its efficacy is denied by others. If used, two or three drops of the solution of strychnia, B P. or I.P., may be injected, and very cautiously repeated at intervals of fifteen or twenty minutes.

Chronic poisoning by chloral.—The long-continued use of chloral in medicinal doses may give rise to skin eruptions of the character noted above, impairment of the cerebral functions, and partial paralysis of the limbs. In some cases, insanity has been attributed to chloral drinking.

Detection.—Organic mixtures containing chloral should be rendered alkaline by caustic potash, distilled, and the vapours tested for chloroform.

Bromal hydrate, the corresponding bromine compound, has a similar action on the system to chloral hydrate, but is a more powerful poison.

Nitro-Glycerine or Glonoine, $CH_5(NO_2)_3O_3$.

This is a heavy, very explosive, oily liquid, almost insoluble in water, but soluble in alcohol, ether, and chloroform. Mixed with siliceous earth, it forms the explosive known as dynamite. Nitro-glycerine is a narcotic poison, acting more powerfully when inhaled in vapour, than when swallowed as a liquid. In some persons, even minute doses cause violent headache. Several cases of poisoning by nitro-glycerine have occurred in Sweden, the symptoms being narcotic in character. In a case mentioned by Taylor, vomiting and purging were also present.

GROUP 4.—NARCOTIC GASES.

Carbon dioxide, Carbonic Acid Gas, CO_2.

This gas is a product of respiration, combustion, and fermentation, and of the decomposition of organic matter. It is also evolved during the decomposition of carbonates by heat, as

in lime-burning; or by acids, as in the chemical preparation of the gas. Poisoning by carbon dioxide is usually accidental. In some countries, however, *e.g.*, France, exposure to the fumes arising from a pan of burning charcoal placed in a room, the door, windows, &c., of which have been tightly closed, is a favourite method of committing suicide. (See also Carbon monoxide.)

Accidental cases may arise from carbon dioxide disengaged in any of the ways mentioned above; for example, from carbon dioxide evolved as a product of (1) **Respiration,** as when they occur in consequence of a number of persons sleeping in a small, badly-ventilated room. (2) **Combustion,** in a similar way to the suicidal cases mentioned above. Accidental cases of this kind have occurred in India (see *Cases* B 32). Under this head also, come cases of poisoning by "choke damp," or carbon dioxide, formed as a product of a coal mine explosion. (3) **Fermentation**; carbon dioxide evolved in this way is liable to accumulate in vats in which fermentation has been conducted, *e.g.*, brewers' vats, rendering descent into the vat, in order to clean it, dangerous to life. (4) **Decomposition of organic matter.** Carbon dioxide thus produced is liable to collect in old wells, pits, vaults, &c., and to give rise to accidents. Descending into pits used for storing grain, which have been for a long time closed, may result in death from carbon dioxide poisoning. A case of this kind occurred at Saharunpore (see *Case* B 33). (5) **Decomposition of carbonates.** Persons sleeping close to a lime-kiln have died of carbon dioxide poisoning; and Taylor mentions a case of accidental poisoning, arising from the use of chalk to neutralize a quantity of nitric acid which by accident had leaked into a room.

Symptoms.—When pure, carbon dioxide causes spasm of the glottis and death from apnœa; diluted, it appears to act as a narcotic poison,[1] causing narcotism, followed by coma and death. The more the gas is diluted, the more gradually it produces its effects. If much diluted, there is at first headache, giddiness, and singing in the ears, and gradual loss of muscular power. Usually the face is livid, and there is palpitation, and hurried respiration. Gradually narcotism supervenes, deepening into coma with stertorous breathing. Sometimes vomiting and convulsions are present.

Toxic percentage.—Considerable difference of opinion exists on the question, what percentage of carbon dioxide present in air may be considered to render it poisonous. When carbon dioxide is simply added to air containing its normal percentage of oxygen, probably eight to ten per cent.—some say more—would be required. When developed at the expense of the oxygen of

[1] It is alleged by some that carbon dioxide is not poisonous, and that its apparently poisonous action on animals is simply due to their being deprived of oxygen.

the air by **respiration**, probably five per cent. would suffice; very much less than this would probably cause distress in most persons, and 2 per cent., it is stated, occasions severe suffering. When developed at the expense of the oxygen of the air, by **combustion**, carbon monoxide is usually at the same time formed, which being more poisonous than carbon dioxide, augments the toxic action of air vitiated in this way. It is very important to note that a candle will continue to burn in air containing a poisonous percentage of carbon dioxide.

Post-mortem appearances.—The face may be pale, or livid and swollen. The tongue is often protruded and grasped by the teeth; sometimes there is froth at the mouth and nostrils. Internally, the appearances are similar to those of death by apnœa. Usually there is much congestion of the brain and its membranes, and of the abdominal viscera.

Treatment.—Remove the patient at once into pure air. Endeavour to restore sensibility by cold affusion and galvanism. Employ artificial respiration, and if there is much congestion, moderate bleeding may be resorted to. Obviously, if a person on descending into a pit or vat is seen to fall immediately insensible from poisoning by carbonic dioxide, to allow others to descend to his rescue is apt to lead only to a useless waste of life. Taylor cites a case where two men lost their lives in this way in attempting to rescue a boy who had fallen into a brewer's vat. Before persons are allowed to descend, the carbon dioxide should be chased out by driving fresh air into the pit or vat; or lime may be thrown down to absorb the gas.

Quantitative estimation.—This may be effected by filling a large narrow-necked vessel of known capacity with the air to be examined, and adding a measured quantity of lime-water, the alkalinity of which has been first ascertained by a standard solution of oxalic acid. The vessel is then tightly closed, well shaken, and allowed to remain at rest for twenty-four hours. After this, the bottle is opened, the fluid poured out, a measured quantity (say equal to half the volume of the fluid originally poured into the bottle) separated; and the loss of alkalinity ascertained by titration as before, with standard oxalic acid solution. The loss of alkalinity of the whole fluid corresponds to the amount of lime converted into carbonate by the carbon dioxide contained in a quantity of the air under examination, equal to the capacity of the vessel, minus the volume of lime-water used.

The quantity of carbon dioxide likely to be present in an enclosed space, *e.g.*, a room, in which the air has been vitiated by respiration or combustion, may be approximately estimated from the following data. Normal air contains on an average four volumes of carbon dioxide per 10,000 of air. Each adult may be considered to give out from a half to six-tenths of a cubic

foot of carbon dioxide per hour.[1] A candle or small lamp gives off about half a cubic foot per hour. If charcoal has been burnt in the room, the amount of carbon dioxide[2] evolved may be approximately inferred from the weight of the residual ash. Roughly, one pound of charcoal corresponds to twenty-nine cubic feet of carbon dioxide, and leaves about half an ounce of ash.

Carbon dioxide is about half as heavy again as air of the same temperature. Like other gases, it expands and becomes lighter as the temperature rises. Gases, however, diffuse into one another, even against gravity. Hence, for a certain time after it has been evolved, hot carbon dioxide will be found in greatest quantity in the upper, and cold carbon dioxide in the lower strata, of a confined portion of air. After a time, however, the gas will have become uniformly distributed by diffusion. After this has taken place, separation by gravity does not occur.

Carbon monoxide, Carbonic oxide, CO.

Carbon monoxide is obtainable by passing carbon dioxide over red-hot charcoal. A certain quantity of it is always formed during the combustion, under ordinary conditions, of charcoal or other carbonaceous fuel, the amount being greatest when the combustion is least active, and *vice versâ*. It is a powerful narcotic poison, much more powerful than carbon dioxide. Death from inhalation of the products of combustion, *e.g.*, the fumes of burning charcoal, is probably in many cases due to carbon monoxide poisoning.

After death from poisoning by carbon monoxide, the blood is found bright-red in colour, not darkened, as in carbon dioxide poisoning. This is held to be due to the carbon monoxide forming, with the hæmoglobin of the blood, a compound of a red colour (carbonic oxide hæmoglobin). It is asserted by some, that this compound is so stable that it cannot be broken up by simple exposure to air or oxygen; and hence, that in poisoning by carbon monoxide, artificial respiration is useless, and transfusion of arterial blood the only remedy. Others deny this, and hold that the compound does break up on exposure of the blood to air.

Coal gas.—The escape of this gas into badly ventilated rooms has frequently given rise to narcotic poisoning. The chief (according to some, the only) poisonous constituent of coal gas is carbon monoxide. The quantity of carbon monoxide present varies in different specimens. Usually the amount present is 5 to 11 per cent., but as much as 22 per cent. has, it is stated, been found. The constituent usually present in largest quantity

[1] As the air gets vitiated the amount given out per hour decreases a little.

[2] A portion of the carbon, however, will probably have become converted into carbon monoxide, which is more poisonous than carbon dioxide.

in coal gas is methane or light carburetted hydrogen, CH_4 (40 to 45 per cent. or more). Methane, even when present in air in quantity sufficient to form an explosive mixture (5½ per cent. or over) appears to exert little or no toxic action. Its presence in air, however, is a source of danger to life from the risk of an explosion, which may cause mechanical injury, or result in poisoning by carbon dioxide.

Sulphuretted Hydrogen, H_2S.

The decomposition of organic matter may result in the production of this gas, directly, when the matter undergoing decomposition contains sulphur, or indirectly, when the decomposition takes place in presence of a soluble sulphate. In the latter "case the sulphate yields a sulphide which, when acted on by carbonic or other acids, evolves H_2S." Accidental poisoning by sulphuretted hydrogen is liable, therefore, to occur from exposure to the emanations from decomposing organic matter, *e.g.*, in cesspools or sewers. This liability is increased, if an acid liquid finds its way into the sewer. It was to this that Dr. Letheby attributed the accident in the Fleet Lane sewer in February, 1861. Agitation also of the liquid in the sewer, &c., favours the evolution of the gas.

Symptoms.—When concentrated, it causes immediate death. When dilute, it gives rise to nausea and the usual symptoms of narcotic poisoning, *e.g.*, headache, giddiness, and laboured respiration, followed by coma. Sometimes delirium and tetanic convulsions are present. When very much diluted, the symptoms are chiefly nausea and abdominal pain, with febrile disturbance.

Post-mortem appearances.—The body exhales an offensive odour, putrefaction is rapid, and the blood is fluid and dark-coloured; there is general congestion of the viscera, and of the right side of the heart. Woodman and Tidy lay stress on the presence of a dirty brown deposit smeared over the lining membrane of the bronchial tubes, as characteristic of death from sulphuretted hydrogen.

Treatment.—Immediate removal into pure air, cold affusions, stimulants, and inhalation as an antidote, of dilute chlorine, as in hydrocyanic poisoning.

Detection.—Free sulphuretted hydrogen is readily recognised by its characteristic odour of rotten eggs, and by its blackening paper moistened with solution of lead acetate. Sulphides—those of the heavy metals excepted—are decomposed by dilute acids, sulphuretted hydrogen being set free.

Sewer gas may contain, in place of sulphuretted hydrogen, the vapour of hydrosulphide of ammonium, which appears to be equally poisonous. Or again, sewer gas may only contain sulphuretted hydrogen in small quantity, and but little carbon

dioxide, but still produce asphyxia in those breathing it, owing to its consisting almost wholly of nitrogen, *i.e.*, of deoxidised air.

Nitrous oxide or Laughing Gas, N_2O.

This is used as an anæsthetic instead of chloroform, and has caused several deaths. Death from inhalation of laughing gas appears to be due to asphyxia; indeed, it has been asserted that the anæsthetic effect of the gas is due to the production of temporary asphyxia, owing to the circulation of non-oxygenated blood, the blood having no power to separate the oxygen contained in this gas.[1]

Carbon disulphide, Bisulphide of Carbon, CS_2.

This liquid, owing to its solvent action on sulphur, caoutchouc, ordinary phosphorus, and other substances, is largely used in certain industries. The vapour of carbon disulphide, from experiments on animals, has been shown to be a narcotic poison, acting very similarly to chloroform. Cases of chronic poisoning by carbon disulphide vapour, have been observed among the workpeople in factories where the liquid is largely used. The symptoms observed in these cases have been a stage of excitement, followed by one of depression. The first stage begins with headache, indigestion and nausea, and creeping sensations, followed by irritability and excitement of the nervous system, which may terminate in mania. In the second stage there is anæsthesia of the skin and mucous membranes, mental debility, and muscular weakness which may culminate in paralysis.

[1] Joylet and Blanche, quoted by Taylor, Manual, p. 445.

CHAPTER XX.

CEREBRAL POISONS—*continued.*

GROUP 5.—VEGETABLE NARCOTICS.

Opium and its Preparations.

Opium.—Afiyún, Afím (*Hind.*), is the inspissated juice obtained by incision from the unripe capsules of the poppy, Papaver somniferum, N.O. Papaveraceæ. It contains meconic acid and a number of alkaloids, those present in largest quantity being narcotine and morphia. Indian opium, as a rule, contains more narcotine than morphia. Other varieties, as a rule, contain more morphia than narcotine. Of these two alkaloids, narcotine has no narcotic properties, and is officinal I.P. as a tonic and antiperiodic. Morphia, on the other hand, is a powerful narcotic. The percentage of morphia in different samples of opium varies from about 2½ to 15 or even 20 per cent. Indian opium often contains under 5, and seldom contains over 9 per cent. of morphia. Other alkaloids present in opium, *e.g.*, codeine, narceine, and papaverine, also possess narcotic properties. Another alkaloid, thebaia, the proportion of which in opium varies, seldom however exceeding 1 per cent., is a convulsant like strychnia, but less powerful.

In India, nearly 40 per cent. of the deaths from poison, reported to the chemical examiners, are due to opium. Such cases in adults are, as a rule, suicidal. In very young children suicide is, of course, excluded, and cases are either accidental or homicidal. The practice of drugging young children with opium, in order to keep them quiet, widely prevalent in India, often results in cases of the first description. So also, but less directly, does the habit of opium eating, cases being often reported, in which children, getting hold of the box in which opium is kept by their parents, eat a quantity and die. Poisoning by opium also appears to be a somewhat common method of infanticide in India.[1] **In England,** according to statistics collected by Blyth,

[1] Harvey (Beng. Med. Leg. Rep. for 1870–72) states that in the three years ending 1872 thirty cases of infanticide by opium poisoning were reported, and in addition thirty-seven cases of alleged infanticide by poison, of which the majority were probably opium cases.

in the five years ending 1880, altogether 1581 deaths were reported from poison. Of these, 643, or 40·7 per cent. were due to opiates. Of the 643 deaths, 160 occurred in infants; two of these were cases of homicide. Of the adult cases, none were homicidal, and about 27 per cent. suicidal. In England, drugging with opium is occasionally adopted for the purpose of facilitating robbery; it is seldom, if ever, used for this purpose in India, its place being taken by dhatura.

Symptoms.—When solid opium has been swallowed, there is usually an **interval** of half an hour to an hour before the symptoms appear. A shorter interval than this is sometimes observed, especially when the drug has been taken in solution on a nearly empty stomach. In other cases, the interval has been several hours in duration. Intoxication appears frequently to delay the appearance of the symptoms; but in one exceptional case, reported by Christison, the appearance of the symptoms was delayed for eighteen hours without any apparent cause. **At first** there may be **slight excitement**; this, however, is soon followed by giddiness and drowsiness, succeeded by **sopor**, gradually deepening into **coma**, with slow stertorous respiration. The breathing gets more and more shallow, and finally death occurs, usually from paralysis of the respiratory muscles. The **face** is usually pale, the lips livid, and the skin bathed in perspiration. All the **secretions** (that of the skin excepted) are more or less completely suspended. **The pulse** may be at first natural or quickened; afterwards it is usually slow, but becomes small, quick, and irregular, as death approaches. An **odour** of opium may be present in the breath. **The pupils** are contracted and insensible to light, but towards the end become widely dilated.

Rare symptoms.—Vomiting and even diarrhœa are sometimes present. Tetanoid convulsions and lockjaw have been observed, more particularly in children; and when morphia has been taken. Guy also includes delirium, anæsthesia, and paralysis, as occasional symptoms In rare cases, the symptoms assume a remittent form, the patient, after several hours' insensibility, recovering consciousness, but relapsing after an interval into coma, terminating fatally (see *Case* B 1).

Unusual modes of administration.—A case is reported of death from application of a poultice to the abdomen containing about one ounce of laudanum.[1] Serious symptoms have arisen from the application of opium to a wound (see *Case* B 34). Death has occurred from the application of morphia to a wound; also from hypodermic injection of morphia, and from the injection of an enema of opium into the rectum. The introduction of opium into the vagina may cause death, and is, I am informed, a not uncommon method of attempting suicide in some parts of India.

[1] Tardieu, quoted by Blyth, p. 288.

Obviously, in cases of this description, there will be no opium odour in the breath.

Diagnosis.—1. **From apoplexy.** Here the chief points of distinction are: (*a*) **The age and appearance of the patient.** Apoplexy generally, but not invariably, attacks the old, and is more common in fat than in thin persons. (*b*) **The history of the case.** In apoplexy, the symptoms, as a rule, come on abruptly; in opium poisoning they advance gradually. (*c*) **The state of the pupils.** In apoplexy, the pupils are dilated, except in apoplexy of the pons varolii, when they are contracted. In opium poisoning, the pupils are contracted, except towards the end of the case, when they become dilated. (*d*) **The odour of opium** in the breath. This, however, may be absent in opium poisoning. (*e*) Convulsions, a bloated face, and impossibility of rousing the patient, are all more in favour of apoplexy, than in favour of opium poisoning. 2. **From uræmic coma.** Here chief reliance would be placed on (*a*) the history of the case; (*b*) the presence in, or absence from, the urine, of matters indicative of disease of the urinary organs; (*c*) the state of the pupils, contraction pointing to opium poisoning; and (*d*) the presence or absence of an odour of opium, in the breath. 3. **From poisoning by other narcotics.** Alcoholic poisoning is sometimes difficult to distinguish from opium poisoning. In the first, the pupils are usually dilated, but may be contracted; and there is often an odour of alcohol, and not of opium, in the breath. In poisoning by carbolic acid, the pupils, as in opium poisoning, are contracted; here, however, the local action of the poison on the lining membrane of the mouth, would most probably serve to distinguish the case. Contracted pupils, also, are present in poisoning by Calabar bean, may be present in aconite poisoning, and also, except during the fits when the pupils are usually dilated, in poisoning by strychnia. The three poisons last mentioned, however, are not narcotic in action.

The quantity of opium required to cause death is specially affected by: 1. **Age.**—Children are extremely sensitive to the action of the drug (see *fatal dose*). 2. **Habit.**—Persons in the habit of taking opium, or its preparations, acquire a resisting power to its action; *e.g.*, a case is on record of an opium eater who was in the habit of swallowing nine ounces of laudanum (tincture of opium) daily; and another of a lady, aged twenty-six, who took ten grains of acetate of morphia three times a day for ten years. 3. **Disease.**—In some diseases, *e.g.*, tetanus, and diseases accompanied by severe pain, large quantities of opium are tolerated. In other affections, *e.g.*, Bright's disease, comparatively small doses may produce serious effects. 4. **Idiosyncrasy.**—Some persons are easily affected by small doses of opium; others are but little affected by large doses. 5. **Quality of the drug.**—As already pointed out, the percentage of morphia in opium varies

greatly. Garrod, however, remarks that, although good opium contains one-tenth of its weight of morphia, morphia is not ten times, but only about four times, as strong as opium. Hence, although ordinary Indian opium probably, as a rule, contains only one-fourth to one-half as much morphia as B.P. medicinal opium, it does not necessarily follow that the latter is from two to four times as strong as the former.

Fatal dose. Children.—Death has been caused in an infant, five days old, by two drops of tincture; in another, nine months old, by four drops of tincture; and in a third case, one grain of Dover's powder, equal to one-tenth of a grain of opium, nearly killed a child, four months old (Taylor [1]). **Adults.**—In the case of these, the smallest fatal doses recorded are: two and a half grains of extract equal to four grains of crude opium (Taylor [2]), and four and a half grains of opium taken along with nine grains of camphor (Christison [3]). Christison also mentions three cases in which elderly persons, suffering from respectively (1) severe catarrh, (2) cough, and (3) asthma, died from doses equal to less than four grains of opium. In one of these cases, the fatal dose was twenty-five drops of the tincture, and in another, fifteen drops of Battley's solution. The case just mentioned excepted, the smallest fatal dose of the tincture on record appears to be two drachms. Blyth, however, points out that the tincture, as usually sold in England, varies greatly in strength. Recovery has been recorded from very large doses of opium. Woodman and Tidy consider that four grains of opium (presumably English medicinal) would in most cases prove a poisonous dose.[4]

Fatal period.—Shortest recorded, forty-five minutes. Usual period nine to ten hours; in rare cases, two to three days. The prognosis is favourable if the patient survives twenty-four hours.

Treatment.—If the poison has been introduced into a wound, &c., remove it as far as possible and wash out the part. If it has been swallowed, give an emetic; and if this fails to act, use the stomach-pump. Keep the patient awake by walking him about, but be careful not to overdo this and so favour syncope. In order to keep the patient roused, use cold affusion, apply sinapisms, and employ flicking with a wet towel. Administer strong infusion of coffee freely. In advanced cases, galvanism and artificial respiration may be required. Many authorities strongly recommend hypodermic injection of atropine. If this is employed, a quarter of a grain of the sulphate (in adults) may be injected, and the dose repeated at intervals, until the pupils begin to dilate. The efficacy of this mode of treatment has been denied. Cases B 35 show that in opium poisoning very large doses of atropine are tolerated. This, to a certain extent,

[1] On Poisons, p. 557.
[2] *Ibid.*, p. 556.
[3] On Poisons, p. 714.
[4] For. Medicine, p. 371.

supports the theory, that atropine is a physiological antidote for opium.

Post-mortem appearances.—Not characteristic. McLeod summarises them as follows in well-marked cases:—"Brain turgid; lungs congested; the head distended with liquid blood; liver and spleen engorged; mucous membrane of the stomach either natural or slightly and uniformly injected."[1]

Opium-eating, &c.—The habit of opium-eating widely prevails in India. Very generally, the crude drug is used. Sometimes, however, as in Rajputana, a watery decoction, known as "*kusoomba*," is employed. Opium-smoking is also much practised, a watery extract of the drug called "*chandul*" being commonly used for this purpose. The question whether opium-eating, smoking, &c., is or is not injurious to health has been hotly debated. There appears, however, to be a pretty general consensus of opinion among medical men, who have actually practised their profession in countries where these habits prevail, that, used in moderation, opium neither tends to injure health nor to shorten life. Abuse of opium, like abuse of alcohol, may cause derangement of digestion, and general impairment of health. Abuse of opium, however, is much less likely than abuse of alcohol, to injure health; and appears to be much less common among opium-eaters, smokers, &c., than abuse of alcohol is among alcohol-drinkers. In persons accustomed to the use of opium, the preliminary stage of excitement, slight or absent in cases of poisoning, is a marked effect of a moderate dose. Chevers gives a case where a man, presumably an opium-eater, previous to committing murder, swallowed, apparently to nerve himself for the deed, so large a quantity of opium that he died a few hours after committing the crime (see *Case* B 36). (For the question of criminal responsibility in such cases, see p. 242.)

Action on animals.—It has been found that large quantities of opium may be given by the mouth to pigeons and other birds, without narcotism being caused. Blyth remarks that the explanation of this is, that the poison is not absorbed, as subcutaneous injection of morphia has been found to act rapidly on all birds hitherto experimented on. In frogs, opium excites tetanus, followed by paralysis of reflex action.

Preparations of opium.—Of the stronger **B.P. and I.P. preparations**, the following quantities correspond to one grain of opium:—Extractum opii, $\frac{1}{2}$ to $\frac{6}{10}$ of a grain; pilula saponis composita, six grains; pilula plumbi cum opio, eight grains; pulvis ipecacuanhæ compositus (Dover's powder), pulvis opii compositus, and emplastrum opii, ten grains; unguentum gallæ cum opio, about $13\frac{1}{2}$ grains; tinctura opii (laudanum), $14\frac{1}{2}$ minims. Extractum opii liquidum, and vinum opii, are both

[1] Beng. Med. Rep. for 1869, p. 109.

slightly stronger than the tincture[1] **Various patent medicines** and quack nostrums also contain opium, among these may be mentioned black drop, an acetic acid solution of opium about four times as strong as the tincture. Battley's liquor opii sedativus has about the same strength as extractum opii liquidum, B P.; and nepenthe, a nearly colourless alcoholic solution of opium, has about the same strength as laudanum. Godfrey's cordial, used as a sedative for children, contains one grain and a half of opium per fluid ounce Other opiate preparations, used for the same purpose, are, Mrs Winslow's soothing-syrup and Dalby's carminative. The first contains about one grain of morphia, with other opium alkaloids, per ounce, and the second, about one-sixth of a grain of opium per ounce.

Morphia.—The symptoms, &c, in poisoning by this alkaloid, or one of its salts, are similar to those of poisoning by opium, except that convulsions are apparently somewhat oftener present. The alkaloid itself is only very sparingly soluble in water, and is not officinal. The two salts of morphia most commonly employed, both of which are officinal, B.P. and I P, are the acetate and the hydrochlorate. Both these are much more soluble in water than morphia; the hydrochlorate being soluble in about sixteen parts of cold water, and less of boiling water, and the acetate more soluble than the hydrochlorate. The medicinal dose of either salt is one-eighth to half a grain. One grain of either may be regarded as a minimum fatal dose for an adult The I.P. contains the following preparations of these salts.—Of the acetate, a solution, strength four grains to the fluid ounce; of the hydrochlorate, a solution of the same strength, also suppositories, half a grain in each, and lozenges, one thirty-sixth of a grain in each.[2]

Chlorodyne contains morphia Blyth[3] gives a formula for Brown's chlorodyne which corresponds to about, in each fluid

[1] Five to six drops of the tincture equal about ten minims The liquid extract and the wine of opium are prepared from extractum opii (one ounce to the pint), while the tincture is prepared from dry opium, an ounce and a half to the pint. One grain of opium is contained also in the following quantities of other B P. and I P preparations —Fifteen grains suppositoria plumbi composita, twenty grains pulvis kino co, twenty-nine minims linimentum opii, forty grains pulvis cretæ aromaticus cum opio, ninety-six minims tinct opii ammoniata, half a fluid ounce tinct. camphoræ co, and about one ounce enema opii. Ten opium lozenges contain one grain of extract of opium

[2] These are similar to the corresponding preparations of the B P of 1864. The B P of 1885 has the following —Of the acetate, a hypodermic injection, strength one grain in ten minims, and a liquor, strength one grain in a hundred fluid-grains Of the hydrochlorate, a liquor, strength one grain in a hundred fluid-grains, and suppositories and lozenges similar to those of the I P, also a tincture of chloroform and morphia containing in each dose of ten minims, one-forty-eighth of a grain of hydrochlorate of morphia, five-eighths of a minim of dilute hydrocyanic acid B P., one minim and a quarter of chloroform, and one-third of a minim of ether

[3] On Poisons, p 273

drachm, four-fifths of a grain of hydrochlorate of morphia, thirty-six minims of chloroform, and 1·2 drops of Scheele's prussic acid.

Poppy capsules, Post (*Hind.*), are narcotic. Syrupus papaveris, B.P. and I. P., prepared from the capsules freed from the seeds, is said to have a strength equal to about two grains of opium per fluid ounce. The B.P. and I.P. also contain an extract prepared from the same, the medicinal dose of which is two to five grains.

Poppy oil expressed from the seeds, Kashkásh-ka tel (*Hind.*), is used largely in India in food, and appears to be inert. Another oil met with in India under the name of poppy oil, unlike poppy-seed oil, is dark brown in colour, and has a strong odour of opium. It appears to be a vegetable oil which has been used for softening old opium; it is employed as an anodyne application, but I have not been able to detect either morphia or meconic acid in the specimens I have examined.

Detection.—Meconic acid in solution gives, with ferric chloride solution, a blood-red colour, not destroyed by dilute hydrochloric acid (distinction from acetates), and not destroyed by mercuric chloride solution (distinction from sulphocyanic acid). **Morphia** in solution gives: (1) With strong nitric acid, an orange colour changing to yellow; (2) With a mixture of bichromate of potassium solution and strong sulphuric acid, a green colour; and (3) with a mixture of starch solution and solution of iodic acid, a blue colour.

From organic mixtures, meconic acid and morphia may be separated as follows:—Digest solid matters cut up into small pieces (or fluid matters concentrated on a water bath to a syrup) with alcohol acidulated with acetic acid, strain through cloth, evaporate the alcoholic tincture nearly to dryness on a water bath, add a little water and a few drops of acetic acid, and filter. To the filtrate, add excess of sub-acetate of lead solution and boil, filter again; the filtrate will now contain the morphia as acetate, and the precipitate the meconic acid as meconate of lead. These are to be separately treated as follows:—(1) The precipitate is to be suspended in water, sulphuretted hydrogen passed through the liquid, the liquid filtered, evaporated to a small bulk, and tested for meconic acid: (2) The morphia filtrate; sulphuretted hydrogen is passed through this until all the lead is thrown down, the liquid is then filtered and concentrated, and morphia extracted from it as in Stas' process (see p. 184), using amylic alcohol or acetic ether as a solvent.[1] As an aid to diagnosis in a case of poisoning, the ferric chloride test for meconic acid, may be applied directly to a small portion of the liquid removed by the stomach-pump.

Failure to detect, &c.—The detection of opium depends on the recognition of morphia and meconic acid, two substances

[1] Morphia is only very sparingly soluble in ether and chloroform.

which form only a fractional part of the crude drug. A very minute dose of opium, however, containing only an infinitesimal quantity of these substances will prove fatal to a young child. In such cases, analysis often fails to detect the presence of the poison. Cases also have often occurred, in which adults have died from the effects of a considerable dose of opium, and yet it has been found impossible to detect the poison after death in the viscera. Among other cases of this kind, Taylor mentions one of a young woman who died in nine hours from a dose of one and a half ounces of laudanum, and yet, in the contents of whose stomach after death, no trace either of meconic acid, or morphia, could be detected. On the other hand, opium has several times been detected in the contents of the stomach of persons poisoned by it, four months or more after death. The explanation of these differences is probably something as follows. When an individual has lived for some time after swallowing the poison, especially in solution, such portion as has not been got rid of by vomiting, becomes absorbed, and distributed through the body, its constituents after absorption, either undergoing change, or becoming so very widely distributed that unless a very large portion of the body is submitted to analysis, a sufficient quantity of them for identification cannot be isolated. On the other hand, if the poison has been taken in the solid form, or death has been rapid, a portion of it, failing to undergo absorption before death, remains in the stomach, and, as opium has a considerable resisting power to putrefaction, its presence may still be detected even if this is far advanced. Finally, it may be pointed out, that the presence of realgar as an adulterant in opium (see p. 153), might lead to complications of evidence in a case of opium-poisoning.

Cannabis sativa.

Cannabis sativa, N. O. Urticaceæ (see Fig. 31), or common hemp, when grown in India, differs so widely in medicinal properties from the same plant grown in Europe, that it formerly was regarded as a distinct species, hence the old name Cannabis indica. It is met with in the bazaars of India in four forms, viz.:—(1) Bháng, siddhí, or sabzí, the dried leaves and small stalks; (2) Ganja, the flowering tops; (3) Charas, the resin which exudes from the leaves and branches—this is often adulterated: I have found thirty to fifty per cent. of mineral matter in specimens; and (4) Majoon, a sweetmeat prepared with hemp. In India, cannabis appears to be seldom, if ever, used for homicidal purposes. Fatal, accidental or suicidal cases have, however, been reported. Cases also have been reported where the drug has, or appears to have, been used for the purpose of facilitating the commission of an offence. Thus Chevers mentions a case

which occurred at Ahmednagar, in which a woman, having first drugged with majoon, a child aged seven, afterwards murdered him for the sake of his ornaments.[1] Harvey reports a case in which charas appears to have been used by a road-poisoner at Umritsur, in order to facilitate theft.[2] A case also is reported by Dr. Cullen, of Hoshungabad, in which majoon was given to a woman and her daughter, "not with the intention of causing

FIG. 31.—CANNABIS SATIVA.

death, but to effect a criminal purpose." In these two females, the symptoms present exactly resembled those of dhatura poisoning, and it would appear that dhatura is sometimes used as an ingredient of majoon.[3] In a case sent to me by the Bombay Police in 1883, some food, alleged to contain cannabis, and to

[1] Med. Jurisp., p. 225. [2] Beng. Med. Leg. Rep., 1870–72, p. 268.
[3] Ainslie speaks of dhatura as a usual constituent of majoon; Honigberger says it is sometimes used as an adulterant of majoon.

have been used for drugging persons in order to facilitate theft, was found to contain a resinous substance, which, when separated and given to a dog, caused distinct narcotic symptoms.

Symptoms.—Cannabis acts on the brain, causing usually excitement, followed by narcotism. During the stage of excitement, the individual is the subject of hallucinations, usually, but not invariably, of a pleasurable, and often of a sexual character. In this stage, the patient may show no outward indications of excitement, or he may be constantly laughing, singing, or talking, or furiously delirious. This stage is followed by one of narcotism, usually with dilated pupils Commonly, there is tingling and numbness of parts of, or over the whole body, or, in severe cases, general anæsthesia may be present.

Detection.—The active principle of cannabis is generally considered to be a resinoid body, cannabin. In addition, it contains a volatile oil, and from recent researches, apparently, also a volatile alkaloid in minute quantity. Authorities differ on the question of the action of these two last-mentioned substances. Warden and Waddell are of opinion, however, from the results of their experiments, that both the oil and a volatile, apparently alkaloidal, substance separable by distillation from an alcoholic extract of the plant, are inert. Cannabis cannot be identified by chemical tests. Its recognition, therefore, in a medico-legal inquiry must depend on the physical characters of the plant, and its physiological action. As regards the first, Dymock describes the leaves as deep-green in colour; they "have long petioles and are digitate, with linear-lanceolate, sharply-serrated leaflets, tapering to a long smooth point." The same author also states, "The flowers form erect clustered spikes, often six to eight inches long; in the drug (ganja) the spikes are compressed, flat, glutinous, and of a brownish-green colour; they have a peculiar narcotic odour" As regards the second, portions of the drug, if available, may be administered to a dog; or food, sweetmeat, &c., supposed to contain cannabis, may be digested with alcohol, the alcoholic solution filtered and concentrated. The resin may be then precipitated by the addition of water, separated and administered to a dog.

Use of cannabis as an intoxicant.—This is widely prevalent in India, the drug being either smoked—ganja-smoking—or swallowed. Insanity in India is often attributed to indulgence in cannabis. According to Baboo Kanny Loll Dey, of 2283 cases admitted in the Bengal Lunatic Asylums during the five years ending 1867, 878 or 38·4 per cent. were attributed to this cause. Apparently, however, many of these were simply cases of temporary intoxication from cannabis. Chevers remarks that "it is a matter of popular notoriety, both in Bengal and the North-West Provinces, that persons intoxicated with ganja are liable to commit acts of homicidal violence." In some cases of homicide

committed, or alleged to have been committed, while under the influence of cannabis, one person only has been attacked (see *Case* B 37 *a*). Usually, however, the victims are numerous, the case assuming the form known as "running amok." Cases of running amok, however, have been reported, in which the criminal has been under the influence of an intoxicant other than cannabis (see *Case* B 36), and also cases in which the criminal does not appear to have been under the influence of any intoxicant (see *Case* B 38). In running-amok cases, whether while under the influence of an intoxicant or not, usually the first individuals injured, are persons with whom the criminal is at enmity (see *Cases* B 36 and 38 *b*). This, however, is not always the case (see *Case* B 38 *a*). Commonly, when acts of homicidal violence are committed while under the influence of an intoxicant, some motive is traceable for the crime. In some cases the motive will, on inquiry, be found to have pre-existed the intoxication; and when this is so, there often appears to be reason to suppose that the intoxicant is taken by the criminal with the object of nerving himself for the deed (see *Cases* B 36 and 37 *a*). In other cases, the motive, such as it is, apparently comes into existence subsequent to the commencement of the intoxication (see *Case* B 37 *b*). As already pointed out, the question of criminal responsibility for acts done while in a state of intoxication, is not affected by the nature of the intoxicating agent. Hence, sects. 85 and 86 of the Indian Penal Code, apply with the same force to cannabis intoxication, as they do to alcoholic intoxication.

CHAPTER XX

CEREBRAL POISONS.—GROUP 6. MYDRIATIC DELIRIANTS.

SOLANACEOUS DELIRIANTS.

Dhatura.

Datura alba and Datura fastuosa, white-flowered and purple-flowered datura, Safed dhatura and Kala dhatura (*Hind*, *&c.*), Umattai (*Tam.*), common in India; and **Datura Stramonium,** or thorn apple, indigenous in England, and officinal B.P. The seeds, leaves, and probably other portions of these plants, contain the poisonous alkaloid daturia or daturine, now regarded as identical with atropine.

Dhatura poisoning is common in India, the seeds being usually employed; a few cases of poisoning by the leaves have, however, been reported. Harvey states that in Bengal, &c., in the three years ending 1872, the seeds were administered in seventy-seven cases, affecting one hundred and twenty-three persons; and the Bombay Analyser's reports for the ten years ending 1885, show that dhatura was detected during that period in seventy cases, affecting one hundred and thirty-eight individuals. In the great majority of cases of dhatura poisoning in India, the motive of administration is facilitation of theft (see *Cases* B 39 and 40). When, in fact, in India, an individual has been first drugged and then robbed, it will usually be found that dhatura has been employed. Sometimes, however, arsenic or cannabis is used. A common form of theft by aid of dhatura is road robbery (see *Cases* B 39); and Dr. W. Center[1] mentions the use in such cases of a hollow pestle, the cavity containing dhatura or arsenic. Inversion of this while pounding grain, &c. with it, introduces the poison into the food without exciting suspicion. Occasionally the motive of administration is other than aid to theft (see *Cases* B 41). It is of course possible that in some cases dhatura is given with homicidal intent. It rarely, however, happens that there is any ground for suspecting this; in fact, there seems to be a wide spread popular belief in India that dhatura is simply an intoxicant, and not a poison. As Harvey remarks, road poisoners sometimes partake with their

[1] 'Indian Medical Gazette' for 1874, p. 116.

victims of the drugged food, which they would hardly do if aware of the danger (see *Case* B 39 *c*). Commonly, where dhatura is used for criminal purposes in India, the poison is mixed with sweetmeats or food, but in exceptional cases the poison seems to have been mixed with tobacco given to the victim to smoke. Suicidal poisoning by dhatura, if it occurs at all, is extremely rare. Accidental cases among children are sometimes met with. *Case* B 42 is an exceptional case of this kind. Lastly, it may be mentioned that dhatura is said to be used in India by vendors of native liquor, for the purpose of conferring additional intoxicating power on their wares. A method said to be followed in Bombay, is to pour the liquor into a vessel which has been first filled with the smoke of burning dhatura seeds.[1]

Symptoms.—These, if a decoction of the seeds has been swallowed, may appear almost immediately. Usually, however, there is an interval, commonly not more than half an hour, between swallowing the poison and first appearance of the symptoms. The first symptom noticed is dryness of the throat: this is followed by giddiness, flushing of the face, and delirium with **widely dilated pupils.** The dryness of the throat increases, and swallowing becomes difficult, the difficulty seeming to depend, at any rate partly, on spasm of the pharyngeal muscles. The voice becomes changed, articulation becomes indistinct, and in one case mentioned by Taylor (from D. Stramonium) the power of speech was lost. The vision becomes indistinct or disordered. **The delirium is peculiar** in character. The patient is restless, often wanders about, talks incoherently, or mutters indistinctly, but at the same time is timid and easily controlled. He goes through various ludicrous movements, appears to grasp at imaginary objects, picks at his clothes or bedding, and often appears to be trying to pull imaginary threads out of the ends of his fingers. These symptoms either gradually disappear, or are succeeded by a **stage of sopor** with subsultus tendinum, deepening into coma sometimes accompanied by convulsions, followed by gradual failure of the heart's action and respiration, and death. Dr. Giraud, in two out of four cases of deep coma, found a remarkable tympanitic condition of the abdomen to be present. If the case tends towards recovery, the sopor passes away, and is succeeded by a stage of **secondary delirium** lasting about six to ten hours, and in character similar to the primary delirium of the first stage. The pupils are widely dilated throughout.

Fatal dose.—This cannot be stated with certainty. Waring, however, writing of the tincture of the seeds (strength 1 to 8), considers twenty drops to be equal in effect to one grain of opium.[2] On this basis, a minimum fatal dose of the seeds

[1] The occasional presence of dhatura in majoon has been already mentioned; see Cannabis.

[2] 'Pharmacopœia of India,' p. 176.

would be about ten to fifteen grains, and a case is reported in which a decoction of 125 D. Stramonium seeds, equal to about sixteen grains, caused the death of an adult in seven hours.[1] The leaves are less active than the seeds. One hundred seeds of D. alba weigh about twenty-one grains; of D. Stramonium, about twelve and a half grains, and of D. fastuosa, about ten grains.

Mortality.—Dr. Giraud (in 1848) met with only one death in fifty-one cases admitted into the Jamsetjee Jejeebhoy Hospital, Bombay; and in the ten years ending 1885, of fifty-nine cases admitted into the same hospital, only two died. This, however, is an exceptionally low death-rate. Dr. Brown, of Lahore, records twenty-one deaths in ninety-two cases. In Harvey's one hundred and twenty-three Bengal cases, twenty deaths were reported; and of the Bo. Analyser's one hundred and thirty-eight cases, twenty-four died. These last three sets of figures give a total of sixty-five deaths in three hundred and fifty-two cases, or just under 18½ per cent.

Post-mortem appearances.—These are usually, wide dilatation of the pupils; congestion of the brain and its membranes, and often also of the lungs and abdominal viscera. The mucous membrane of the stomach and intestines may be found congested, and patches of extravasated blood have been met with in the large intestine. Seeds, or fragments of the seeds (see *Detection*), may be found in the contents of the stomach or intestines.

Treatment.—Administer emetics, or use the stomach-pump; and treat the symptoms, as they arise, on general principles, *e.g.*, if the pulse is feeble, and the skin cold, give stimulants; if narcotism is present, use cold affusion, &c., as in opium poisoning; employ artificial respiration if necessary. Opium may be given if there is much excitement. Opium, however, is less efficient as an antidote in atropine poisoning, than atropine in opium poisoning. Lauder Brunton recommends the cautious administration of physostigma; and Ringer and others advise, in atropine or stramonium poisoning, administration of pilocarpine nitrate in quarter to half grain doses.

Detection.—**Dhatura seeds** are ear-shaped, and to a certain extent **resemble capsicum seeds.** In both, the testa is, under the microscope, seen to be covered with convoluted ridges. Whole seeds or large fragments may readily be differentiated as follows:—(1) Dhatura seeds are **double-edged** at the convex border; capsicum seeds are not; (2) Capsicum seeds have a sharp pungent taste; dhatura seeds are feebly bitter, but **not pungent**; (3) If the seeds are laid on the flat, and divided horizontally, the **embryo** of a dhatura seed is seen to **differ in shape** from that of a capsicum seed (see Figs. 33 and 34);

[1] Taylor on Poisons, p. 774.

(4) A little solution of **iodine** applied to the section, **gives** (if the seeds are tolerably fresh) **a blue tint** at a spot near the hilum of the seed, if it be a dhatura seed, but not if the seed is a capsicum seed. **From organic mixtures**, or the powdered seeds, the alkaloid may be separated by Stas' process, using ether as a solvent. It will be found when dissolved in a little

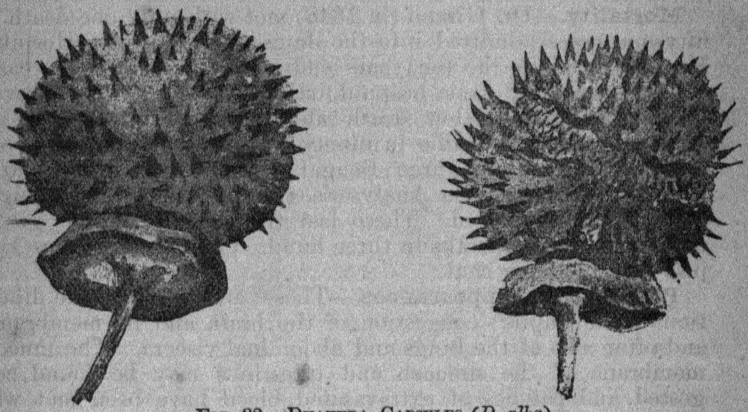


FIG. 32.—DHATURA CAPSULES (*D. alba*).

very dilute acid (1) to respond to the group tests for alkaloids, and (2) when applied to the conjunctiva, to cause dilatation of the pupil. There are no special chemical tests for daturine (or atropine); the physiological test (action on the pupil) is, however, extremely delicate. In man, an atropine solution of 1 to 120 commences to act in six or seven minutes, and its effects

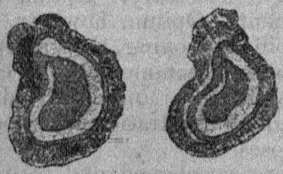


FIG. 33.—SECTIONS OF DHATURA SEEDS (*enlarged*).

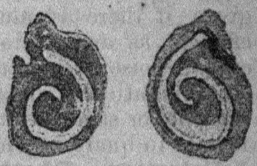


FIG. 34.—SECTIONS OF CAPSICUM SEEDS (*enlarged*).

continue more or less for several days. A solution even of 1 to 48,000 will slowly cause dilatation. Herbivora, and especially the rodents, are much less sensitive than man to the action of atropine.

Belladonna.

All parts of the **Atropa belladonna**, or deadly nightshade, indigenous in England, contain atropine. Cases of poisoning

by belladonna are occasionally met with in England, and are usually accidental, arising either from eating the berries in ignorance of their poisonous nature, or from mistakes in dispensing or using medicinal preparations. Accidental cases of this last description have been reported in India.

The Symptoms are similar to those of dhatura poisoning; in several cases a scarlet rash on the skin has been observed. Recovery is frequent; in over sixty cases of belladonna and atropine poisoning tabulated by Woodman and Tidy, there were only fourteen deaths. Death has been caused by a few of the berries; and two of the berries, and three grains of the extract, have each caused bad symptoms. Death also has resulted from the application of atropine to a blistered surface.

Ordinary Medicinal doses are, of atropine, one hundredth to one twenty-fifth part of a grain; of extract of belladonna, one quarter to one grain; and of the tincture (strength, one of the leaves to twenty of proof spirit), five to twenty minims.

The post-mortem appearances and treatment are the same as in poisoning by dhatura.

Detection.—The flowers have a bell-shaped corolla about one inch in length, dull reddish purple in colour, and pale green at the base. The berries are rounded, about three-quarters of an inch in diameter, purple black and shining. The seeds are small, about one-tenth of an inch in diameter, and studded with projections. Organic mixtures may be subjected to Stas' process as described under detection of dhatura.

Hyoscyamus or Henbane.

Hyoscyamus niger and H. alba.—The leaves of the former are officinal in the British and Indian Pharmacopœias. Hyoscyamus seeds, apparently seeds of H. alba, are met with in the Indian bazaars, under the names of Khorasani-ajwan (*Hind.*), Khorasani-owa (*Bomb.*), and Khorasani-omam (*Tam.*). The two plants named, and probably other species of hyoscyamus contain a poisonous alkaloid, hyoscyamine, the action of which is similar to that of atropine, but weaker in degree. Ordinary medicinal doses are—of the extract (of the leaves) five to ten grains; and of the tincture (strength, one of the leaves to eight of proof spirit), half a drachm to a drachm. A few (not Indian) cases of poisoning by hyoscyamus have been reported. In one, a woman suffered severely, but recovered from a dose of eleven drachms of the tincture; and in another, six adults, of whom one died, were poisoned by eating the roots in mistake for parsnips. In a third, two boys, one of whom died, were poisoned by the seeds. In India, the seeds are used in native medicine, and according to Birdwood, are frequently employed criminally in

Bombay. I, however, have not met with any criminal cases of hyoscyamus poisoning. Treatment as in dhatura poisoning.

Hyoscyamus insanus.—Chevers[1] mentions that this species is common in Beloochistan, where it is known as Kohi-bhang, or mountain hemp, and Waring states that it is well known to be powerfully poisonous, and is sometimes smoked in small quantities as an intoxicant, by debauched faquirs.

Detection.—Dymock describes the bazaar seed as "reniform, laterally compressed, equal in size to that of H. niger,[2] of a light brown or grey colour; the testa is finely reticulated. The albumen is oily. The embryo curved like the figure 9, the tail of the nine being represented by the radicle. The taste is oily, bitter, and acrid." Hyoscyamine applied to the conjunctiva dilates the pupil, and may be recognised, but not distinguished from atropine, by this property. It may be separated from organic mixtures by Stas' process as for atropine.

Solanine.

This is a poisonous alkaloid contained in plants belonging to the genus solanum, N. O. Solanaceæ. A few cases of poisoning by plants containing solanine are on record. In many of the cases there have been thirst, vomiting, and purging. Usually severe nervous symptoms are present, *e.g.*, convulsions, delirium, and coma. Dilatation of the pupils has also been reported, but, according to Lauder Brunton, solanine is entirely without action on the pupil. The following species may be specially mentioned:—

Solanum dulcamara, bitter-sweet, or woody nightshade.—This, a common hedge plant in England, is officinal in the British and Indian Pharmacopœias. A child æt. four is said to have died from eating only two of the berries. According to Dymock, the dried fruit is imported from Persia into Bombay, where it is known under the name of Anab-es-salib.

Solanum nigrum, or garden nightshade, Makoi (*Hind.*), Kámuni, Ghati (*Bomb.*), Manattak-kali (*Tam.*), Kakmachi (*Beng.*).—This is found in gardens in England, and is common in India. Woodman and Tidy cite two cases (both in Europe) of poisoning of children by the berries; in one, two were poisoned and one died; in the other, three children, all of whom died, were poisoned.

Solanum tuberosum, or potato.—A fatal case in a girl æt. fourteen from eating the berries of the potato is reported; and another in which four persons, all of whom recovered, were poisoned by eating potatoes which had commenced to germinate. Mature potato tubers, however, contain only a trace of solanine.

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[1] Med. Jurisp. p. 276.

[2] About one-eighteenth of an inch in diameter, and weighing one hundred and twenty to the grain (Guy).

NON-SOLANACEOUS DELIRIANTS.

Wormseed.

The unexpanded flower-heads of **Artemisia maritima**, N. O. Compositæ, Kirmani owa (*Bomb.*), used in medicine as a vermifuge, contain 1½ to 2 per cent. of santonin, a crystalline principle to which their activity is due. Santonin is the anhydride of santonic acid, and is administered medicinally in doses of two to five grains. Large doses of wormseed or of santonin give rise to symptoms of irritant poisoning, accompanied by delirium or convulsions, followed by stupor. The pupils are dilated, and a peculiar disturbance of vision, owing to which everything appears at first bluish, and afterwards yellow or greenish yellow, is a constant symptom. Taylor [1] quotes a case where 155 grains of wormseed proved fatal to a girl aged ten; and Chevers [2] mentions two cases, in each of which three grains of santonin caused serious symptoms in children. Treatment, general as for cerebral poisons.

Detection.—From organic mixtures santonin may be extracted by a process similar to that employed for plumbagin (see p. 219), using chloroform instead of ether as a solvent. When dissolved in slightly diluted sulphuric acid, and the solution warmed, a red colour passing into purple, and finally into brown, is produced by the addition of ferric chloride solution.

Wormwood.

The leaves and tops of **Artemisia Absinthium**, N. O. Compositæ, and probably of other species of artemisia, contain a bitter principle (absinthin) and a volatile oil. The latter is a narcotic poison, causing stupor, convulsions, and dilated pupils. In one case half an ounce of the oil caused very severe symptoms in a male adult. Absinthe, a French liqueur, contains oil of wormwood, and when taken in excess gives rise to loss of intellect, paralysis, and epileptiform convulsions, combined with the usual effects of chronic alcoholic poisoning.

Tansy.

Tansy, Tanacetum vulgare, N. O. Compositæ, contains a volatile oil possessing apparently a similar action on the system to oil of wormwood. Woodman and Tidy [3] mention two cases of poisoning by tansy, taken with the object of causing abortion. In both cases coma supervened, but in neither was miscarriage produced. One of the two terminated fatally.

Coriaria myrtifolia.

A few cases of poisoning by the berries and leaves of this plant—a native of Europe—have been reported, the symptoms being coma, convulsions, and dilated pupils. In one case an adult died in twenty-four hours from eating fifteen of

[1] On Poisons, p. 682. [2] Med. Jurisp., p. 283.
[3] 'Forensic Medicine,' p. 451.

the berries. In another an adult died in four hours from swallowing an infusion of senna leaves that had been adulterated with the leaves of this plant;[1] and Taylor[2] mentions a case where a whole family in France was poisoned by eating snails, that had been fed on the leaves and young shoots.

Camphor.

Common or officinal, or Laurel camphor, Kafur (*Hind.*), Karuppuram (*Tam.*), Kapur (*Bomb.*), Karpur (*Beng.*), is a volatile crystalline substance obtained from the Cinnamomum Camphora or Laurus Camphora, N. O. Lauraceæ. Its chemical composition is represented by the formula $C_{10}H_{16}O$. Another variety, **Borneo camphor,** from the Dryobalanops aromatica, N. O. Dipterocarpeæ, has the composition $C_{10}H_{18}O$. Camphor is administered medicinally in doses of one to ten grains. In large doses it acts as a poison, causing excitement and delirium, with dilated pupils and sometimes convulsions. Several cases of poisoning by camphor, nearly all of them non-fatal, are on record. In one case thirty grains caused furious delirium in an adult, and in another twenty grains, swallowed in solution by an adult, caused severe symptoms. Recovery has been reported from a dose of 160 grains, and in another case from a dose of 270 grains.

Poisonous mushrooms.

Certain species of mushrooms are non-poisonous and are used as articles of food. Others are poisonous, and cases of accidental poisoning occasionally occur, from one of the poisonous being mistaken for an edible variety. Poisonous mushrooms, unlike non-poisonous mushrooms, have a bitter, astringent. acrid, or salt taste, and on section and exposure change colour, a brown, green or blue tint developing on the cut surface. In mushroom poisoning the symptoms present may be those of irritant poisoning, or those of cerebral poisoning, or both sets of symptoms may be present. Prominent cerebral symptoms in mushroom poisoning are, excitement and intoxication, convulsions, delirium, and stupor, with dilated, or in some cases contracted pupils. Of the better known poisonous varieties, one, the Amanita muscaria or fly-blown agaric, appears to owe its activity partly to an undiscovered substance destroyed at the temperature of boiling water, and partly to an alkaloid called muscarine. Muscarine taken internally causes contraction of the pupils:[3] hence this condition is present in cases of poisoning by Amanita muscaria.[4] Atropine appears to be to a great extent antagonistic in its action to muscarine, and is recommended as an antidote. A curious fact about poisoning by Amanita muscaria, is that it renders the urine intoxicating; and in Kamtchatka, where this fungus is used as an intoxicant, individuals are in the habit of drinking their urine so as to renew the intoxicant effect. Cases of poisoning have also been reported in Europe from the common morelle. The poison of this variety appears to be soluble in boiling water, and volatile, and to disap-

[1] Woodman and Tidy, p. 293.
[2] On Poisons, p. 169.
[3] Lauder Brunton's Pharmacology, p. 187.
[4] Woodman and Tidy, p. 306.

pear when the morelles are cooked or dried. **Poisonous mushrooms** are to be met with **in India.** Chevers mentions a case of mushroom poisoning which occurred at Jessore in 1853, and Dr. Kirtikar informs me that, although the poisonous varieties growing in India are not many, he has been able to identify the following: Agaricus procerus, Agaricus albus, Agaricus dryophilus, Boletus atrofulvus, and some varieties of Boletus nitidus crocatus, especially the variety growing on the peepul tree. Of these the first three, viz., Agaricus procerus, albus, and dryophilus, give rise to mixed irritant and cerebral symptoms. Boletus atrofulvus, from a case observed by Dr. Kirtikar, seems to act chiefly as a cerebral poison. Dr. Kirtikar also informs me that he once met with a case in which the external application of the peepul variety of Boletus nitidus, as an anodyne, was followed by a good deal of vesication of the skin, with eczematous inflammation in some places.

Dr. Paxton, of Chichester,[1] points out that examination under the microscope of portions of a suspected mushroom, or of the contents of the stomach in a case of suspected mushroom poisoning, may be employed in order to determine whether or no the mushroom concerned is poisonous. He considers that the discovery of pink irregular spores, or of rusty brown irregular spores, or of round white prickly spores, is strong evidence of the mushroom being poisonous; while plain round spores may belong to a poisonous or to a harmless variety.

Treatment.—Emetics followed by stimulants, warmth to the surface, and hypodermic injection of atropine in one-fiftieth grain doses.

APPENDIX TO CEREBRAL POISONS.

Poisonous Food-Grains.

It has already been mentioned (p. 216), that various cereals affected with ergot become poisonous. Cases of poisoning also have occurred from, or been attributed to, the following:—

Lolium temulentum, Darnel, or Bearded darnel; Mostaki (Punj.), Mochni (N.W.P.).—A few cases of poisoning, mostly non-fatal, have occurred both in India and in Europe, due to the consumption of bread, &c., made from grain containing darnel seeds. The symptoms of darnel poisoning are chiefly giddiness with tremors of the muscles, and dilated pupils followed by sopor. Irritant symptoms may also be present. Christison mentions three European cases of mass poisoning by darnel, affecting respectively eighty, seventy-four, and forty persons, all of whom seem to have recovered. Similar cases have been reported as occurring at hill-stations in the Punjaub and N.W. Provinces. The precise nature of the poisonous principle of darnel is unknown.

[1] See Woodman and Tidy, p. 306.

Identification.—According to Hassall, the starch granules of darnel are polygonal, like those of rice, but much smaller. The structure of the testa also, in the main resembles that of rice, differing however in the fact, that in darnel the outer coat consists of a single layer of broad cells disposed transversely, and not of narrow transverse fibres as in rice.

Paspalum scrobiculatum, Kodra or Harik.—Cases of poisoning are occasionally met with in India, arising from the consumption of this grain as an article of food. The symptoms of kodra poisoning are very similar to those of poisoning by darnel, viz.: tremors and twitchings of the muscles, giddiness and seeming intoxication, with impaired vision followed by sopor, and accompanied in some cases by irritant symptoms. Kodra poisoning occasionally ends fatally: thus in a case reported to the Bombay Chemical Analyser, from Godhra, in 1879–80, four persons, viz., a man and three children, were poisoned by eating bread made from kodra flour, and one of the children died. Kodra appears to be only occasionally poisonous; according to popular belief, in fact, it is supposed that there are two varieties of the grain, a sweet and a bitter variety (Gora harik and Majara harik), of which the latter alone is poisonous. As is the case with darnel, the precise nature of the poisonous principle present in kodra is unknown. So like however are the symptoms of kodra to those of darnel poisoning, that it has been suggested that so-called kodra poisoning is really darnel poisoning, arising from accidental mixture of darnel with the grain.

Lathyrus sativus, Kesarí dál or Láng.—This pulse is used by the poorer classes in some parts of India as an article of food. Its continued consumption is said to give rise to a kind of chronic poisoning, characterised by rheumatic pains, followed by paralysis of the lower extremities. According to Dr. W. Kirk,[1] paralysis of the lower limbs, resulting from the use of kesarí dál as an article of food, is extremely common in Sind.

Certain of the **poisons** already described under **Vegetable Irritants** act also on the brain. Thus stupor or insensibility with dilated pupils has been observed in poisoning by Tylophora fasciculata, Daphne Mezereum, the Arums, Laburnum, and Yew. Cerebral symptoms are also present in cases of poisoning by Cocculus indicus, and the fruit of Terminalia bellerica. Lastly, it may be mentioned that cases have been met with in India (chiefly among children) which tend to show that the kernels of the fully developed seeds of star-anise, Illicium anisatum, possess a narcotic action.

[1] Chevers' Med. Jurisp., p. 305.

CHAPTER XXII.

SPINAL POISONS.—GROUP 1. CENTRAL POISONS.

SPINAL POISONS.

UNDER this head may be grouped a few very active neurotic poisons, the main action of which is exerted on the spinal cord, or on the peripheral extremities or trunks of the cerebro-spinal nerves. This action may be stimulant in character or the reverse. If stimulant, the result may be production of muscular spasm (as in strychnia poisoning), or tingling of the surface, as in aconite poisoning. If the reverse, paralysis, or loss of sensation results. Cerebral symptoms are, as a rule, either absent or slight, and death usually occurs by asphyxia, due to arrest of the movements of respiration. This arrest may (as in strychnia poisoning) be the result of spasm, but is more commonly due to paralysis. Some poisons of this order, viz. Calabar bean and aconite, are cardiac as well as spinal poisons, and slow the action of the heart. In poisoning by these death may occur by syncope.

The general indications of treatment may be carried out in spinal poisoning as follows: (1) Elimination, by giving emetics or using the stomach-pump; (2) Prevention of action by administration of animal charcoal, or of gallic acid, tannin or decoctions containing tannin; (3) Counteraction of effects, by treatment of the symptoms as they arise, *e.g.*, administration of stimulants to counteract depression, employment of artificial respiration, &c. Certain drugs more or less antagonise the action of some of the poisons of this order and are recommended for use as physiological antidotes; thus, in strychnia poisoning, inhalation of chloroform is specially indicated; and in poisoning by aconite, administration of atropine, or, if there is much cardiac depression, of digitalis, is advised.

Spinal poisons may conveniently be divided into (1) those which specially affect the cord, or central poisons, and (2) those which primarily affect the peripheral extremities or trunks of the nerves, or peripheral poisons. The central spinal poisons include strychnia, calabar bean, and gelsemium; of these the first excites, and the other two paralyse the cord. The peripheral spinal poisons include curara and conia, acting specially on the motor nerves, and aconite, acting specially on the sensory nerves.

GROUP 1.—CENTRAL POISONS.

Strychnia.

Certain plants belonging to the genus Strychnos of the N. O. Loganiaceæ,[1] contain **strychnia,** and another poisonous alkaloid of similar action but milder in degree, named **brucia.** The two alkaloids are contained in nux vomica, in combination with **strychnic** or **igasuric acid,** probably identical with malic acid. Strychnia and brucia have been found present in :—

1. **Strychnos Nux vomica,** Kuchila (*Hind.*), Kajra (*Bomb.*), Ettikkottai (*Tam.*). The seeds of this are officinal, and the bark is met with in commerce under the name of "false angostura bark."

2. **Strychnos Ignatii,** Papita (*Hind.* and *Bomb.*), Kayappankottai (*Tam.*). The seeds of this are known as Faba amara and St. Ignatius' beans.

3. **Strychnos colubrina,** Snake-wood, Naga-musadi (*Tel.*), Modira-caniram (*Mal.*), Kuchila-lata (*Hind.*), Goagari-lakri (*Bomb.*), Kajarwel (*Mar.*), and

4. **Strychnos Tieute,** the upas tree of Java.

Strychnos toxifera belonging to the same genus is believed to be the chief source of curara, which see. Several species of strychnos are non-poisonous: of these the most important is the Strychnos potatorum, Nirmali (*Hind.*), Tetrankottai (*Tam.*), Nivali (*Mar.*), the seeds of which are used under the name of clearing nuts, for clearing muddy water.

In India, poisoning by nux vomica is occasionally met with, the cases being generally suicidal or accidental. Among the causes leading to accidental poisoning may be mentioned, (*a*) The practice of nux vomica eating, which to a certain extent prevails in some parts of India (see *post*, Influence of habit), and (*b*) The substitution of nux vomica or kuchila bark for other barks, notably for kurchi or holarrhena antidysenterica bark, a drug in common medicinal use in India as a tonic and antiperiodic for children, and as an astringment in dysentery. In a case which occurred in Calcutta in 1882, the death of a child was traced to this substitution, and in a subsequent case, on a vendor's stock of holarrhena bark being seized, about one-fourth of it was found to consist of nux vomica bark. Waddell, in fact, suggests that this substitu-

[1] Strychnia appears also to be present in certain parasitic plants of the N. O. Loranthaceæ, growing on nux vomica trees. One of these, believed to be Viscum monoicum, *vern.* Kuchila ke molung, is found in the neighbourhood of Cuttack. Chevers mentions a case of poisoning by the pounded leaves of this plant, in which the symptoms were precisely those of strychnia poisoning. A poisonous species of loranthus has also been described as growing on nux vomica trees in the Neilgherries, the Tamul name of which is said to be Poulourivi.

tion may partly account for the great mortality among infants and children, yearly reported from tetanus in Calcutta.[1] **Poisoning by the alkaloid strychnia,** formerly rare in India, has of late years, at any rate in the Bombay Presidency, become more frequent, owing to the greater ease with which the alkaloid can be obtained. In the Bombay Presidency, strychnia powders have of late years been largely supplied to the police for the purpose of destroying dogs; and several cases of poisoning of human beings by strychnia have been reported, in which the poison was obtained from this source.

Action, Symptoms, &c.—Strychnia acts as a direct stimulant to the spinal cord, causing tetanic spasms, and death either from **asphyxia,** due to spasm of the muscles of respiration; or from **collapse,** occurring in the interval between the spasms. When swallowed (except when taken in the form of a pill) the first thing noticed is an intense bitter taste: this is frequently but not invariably succeeded by a feeling of suffocation and want of air. Twitchings and cramps follow, rapidly developing into intensely painful tetanic spasms, affecting nearly all the muscles of the body. During the spasms the body frequently becomes rigid, and arched so as to rest only on the head and the heels (opisthotonos). During the fits of spasm also, the pupils are usually dilated, and the features drawn into a grin (risus sardonicus). The fits of tetanic spasm alternate with intervals of muscular relaxation, the relaxation being as a rule complete. As the case progresses towards a fatal termination, the intervals between the spasms become shorter and shorter in duration. There is no narcotism, but insensibility from exhaustion may occur before death.

Fatal period, &c.—In poisoning by strychnia, the symptoms usually appear in five to twenty minutes; in poisoning by nux vomica the appearance of the symptoms is less rapid, and in one fatal case their appearance was delayed for two hours.[2] Death may occur in ten minutes or be delayed for five or six hours. Of thirty-five cases collected by Woodman and Tidy,[3] nineteen died in an hour or less, and eleven more in one to three and a half hours.

Diagnosis from disease.—The chief points of distinction between strychnia poisoning and **tetanus,** the only disease likely to be mistaken for it, are: (1) In poisoning, the symptoms come on suddenly, rapidly become severe, and soon end either in death or recovery; in tetanus as a rule, the reverse is the case. Chevers, however, mentions an exceptional case of sudden accession of tetanus, and death in the first spasm (see *Case* B 43). (2) In tetanus the muscles of the lower jaw are early affected, stiffness of the lower jaw being often the first prominent symptom; in poisoning, the muscles of the lower jaw are the last to be affected.

[1] 'Indian Medical Gazette,' March 1885.

[2] Taylor's Manual, p. 172.

[3] For. Medicine, p. 330.

(3) In poisoning, as a rule, the muscles during the intervals are completely relaxed: this is not so in tetanus.

Treatment.—Administer emetics or use the stomach-pump, chloroforming the patient so as to allow of the introduction of the tube. Give animal charcoal or infusions containing tannin. Administer chloral, or still better, keep the patient under the influence of chloroform.

Post-mortem appearances.—Not characteristic; as a rule the body is relaxed at death, sometimes however it remains rigid, the rigidity continuing for a very long period. The brain, spinal cord, and lungs are usually found congested; the heart may be empty or full.

Fatal dose, &c.—(*a*) **Of strychnia.** A medicinal dose of this is one-thirtieth to one-twentieth of a grain. Taylor[1] estimates the fatal dose for an adult at half a grain to two grains Some persons appear to be specially sensitive to the action of strychnia, and two cases are on record in which respectively one-twelfth, and one-sixth of a grain, caused alarming symptoms. In both these cases the subjects were adult females. The smallest quantities which have caused death, are one-sixteenth of a grain in a child two to three years old, and half a grain of the sulphate in an adult.[2] Recovery has been recorded from doses of ten to twenty, and even forty grains of the alkaloid.[3] (*b*) **Of nux vomica.** Nux vomica seeds contain about a quarter to half per cent. of strychnia, and St. Ignatius' beans about one and a half per cent.[4] The medicinal dose of powdered nux vomica seeds is two to three grains. In one case thirty grains of powdered nux vomica seeds (equal to about one full-sized seed), taken in two doses of fifteen grains each, caused the death of a girl aged ten; and in each of two cases death was caused by fifty grains.[5] It should be noted that, owing to the insolubility of the testa, **whole** nux vomica **seeds** may be swallowed, and pass through the body, without giving rise to symptoms of poisoning.

Influence of habit.—Many authorities state that in different parts of India, nux vomica is habitually eaten (like arsenic) as a stimulant and aphrodisiac. Baker, quoted by Chevers,[6] states that those who practise this habit, begin with one-eighth of a grain of the seed, gradually increasing the dose to about twenty grains. If this is so, the inference of course is, that habit tends to confer on the system a resisting power to the action of strychnia. It is, however, undoubtedly the case that small doses of strychnia repeated at short intervals tend to exert a cumulative effect. Lauder Brunton, in fact, states that strychnia is a cumulative poison, and points out that this effect is due to the slowness with which it is excreted, one effect produced by it being

[1] On Poisons, p. 713.
[2] Case of Dr. Warner, *ibid.*, p. 712.
[3] Woodman and Tidy, p. 330.
[4] Pharmacographia, pp. 430, 433.
[5] Taylor on Poisons, p. 695.
[6] Med. Jurisp., p. 241.

to contract the renal vessels, and thus interfere with its own elimination.

Unusual Cases.—Harley[1] records a case where an infant at the breast suffered from symptoms of strychnia poisoning, the result of the medicinal administration of strychnia to the mother, who remained unaffected. Dr. Chatterjee[2] met with a non-fatal case, resulting from the introduction, by a quack, of a pulp made from nux vomica seeds into a wound. Blyth[3] mentions a case of attempted suicide by a young woman, who took about one and a half grains of strychnia, and two ounces of laudanum. Severe symptoms of narcotic poisoning followed, but no symptoms of strychnia poisoning showed themselves until eight hours afterwards.

Preparations: (1) **Officinal.**—The following preparations of nux vomica are contained in the I.P. :—

—	Strength.	Medicinal Dose.
Infusion . . .	1 to 48	½ oz. to 1 oz.
Tincture . . .	2 ozs. to 1 pint	5 to 20 drops
Extract . . .	1½ oz. equals about 1 lb. of the seeds	¼ of a grain to 2 grains

The I.P. also contains a solution of strychnia; strength four grains to one ounce, or about 1 to 109. With the exception of the infusion, the above preparations are similar to those of the B.P. of 1867. The strength of the preparations of the new B.P. (of 1885) is defined as follows:—The extract is to be so made as to contain 15 per cent. of nux vomica alkaloids; and the tincture, made from the extract, is to contain one grain of nux vomica alkaloids per ounce. The solution of strychnia (liquor strychninæ hydrochloratis) has a strength of about 1 to 100, instead of 1 to 109 as in the B.P. of 1867. (2) **Non-officinal.**—Various vermin-killers sold in England contain strychnia. Blyth mentions the following:—**Miller's rat powder,** a mixture of one ounce of nux vomica to one pound of oatmeal. **Battle's vermin-killer,** a mixture of strychnia with flour and Prussian blue, containing about 7·7 per cent. of strychnia, and **Butler's vermin-killer,** a mixture of strychnia with flour and soot, or sometimes with Prussian blue, containing from about 3⅓ to 5 per cent. of strychnia.

Identification.—**Nux vomica seeds** are in shape nearly circular discs, slightly concave on one side and convex on the other, about one inch in diameter, by about a quarter of an inch thick. In colour, they are light greyish, and have on the surface a silky appearance, due to their being thickly covered with

[1] Woodman and Tidy, p. 330. [2] Ind. Med. Gaz., 1872, p. 251.
[3] On Poisons, p. 313.

short hairs. They are very tough and horny, and have an intensely bitter taste. **St. Ignatius' beans** are about one and one-fifth of an inch in length, ovoid, but presenting three to five flattened surfaces due to mutual pressure. "In the fresh state they are covered with silvery adpressed hairs; portions of a shaggy brown epidermis are here and there perceptible on those found in commerce, but in the majority the seed shows the dull grey granular surface of the albumen itself."[1] **Nux vomica bark** occurs in quilled twisted pieces, an inch or less in diameter, thin, light-brown in colour, and marked on the outer surface by numerous small light-coloured elliptic corky warts. Its inner surface is turned red by nitric acid:[2] this distinguishes it both from angostura or cusparia bark, and from holarrhena antidysenterica bark; the latter also is much thicker, and is marked on the external surface by scars of exfoliation.

Detection of Strychnia.—Strychnia occurs in commerce as a white powder, or in white rectangular prismatic crystals, but may be obtained in other crystalline forms, *e.g.*, hexagonal prisms, octahedra, or forms derived from the octahedron. It is freely soluble in chloroform (1 to 7 or 8), less soluble in alcohol (about 1 to 100), and still less soluble in ether. It is only very sparingly soluble in pure water (about 1 to 7000 of cold water), but readily dissolves in dilute acids. Its solution in faintly acidulated water has an intensely bitter taste, and is precipitated by the alkaloidal group reagents (see p. 185). The special tests for strychnia are:

1. **The colour test.**—This depends on the fact that a play of colours—viz., blue, rapidly changing to violet, and then slowly to purple, and lastly, to red—is produced when strychnia is subjected to the action of nascent oxygen. To apply the test, stir a minute quantity of strychnia with a drop or two of strong sulphuric acid; the strychnia dissolves if pure, without change of colour. Then cautiously add a minute quantity of manganese dioxide, or lead dioxide, when the play of colours appears. Potassium permanganate, or potassium dichromate, may be used instead of manganese or lead dioxide, but are not so suitable; or the test as proposed by Letheby, may be conducted galvanically. In this modification, the strychnia dissolved in a drop or two of strong sulphuric acid, is placed on a piece of platinum foil connected with the positive pole of a galvanic battery; on touching the liquid with the negative pole, the play of colours appears. It has been objected that various substances, *e.g.*, pyroxanthin, piperine, salicin, bile, &c., become coloured when treated with strong sulphuric acid. With these substances, however, the colour appears directly the acid is added, while with strychnia no colour appears until after the addition of the manganese dioxide, or other oxygen yielding substance.

[1] Pharmacographia, p. 388. [2] Owing to the presence of brucia (see p. 280).

Aniline salts, Woodman and Tidy point out, are not coloured by sulphuric acid only, but give a play of colours when manganese or lead dioxide is added; with aniline salts, however, the colours are first "green, then a very persistent blue, then black."

2. The physiological test.—This consists in administering, preferably by subcutaneous injection, a little of the suspected alkaloid, to a small animal, *e.g.*, a frog, and observing whether or no tetanic symptoms are produced. Substances other than strychnia, however, induce tetanic symptoms: hence the physiological test is only useful as a negative test, to prove the absence of strychnia; or, as a confirmatory test. If, however, definite chemical evidence of the presence of strychnia has been obtained, the physiological test is quite unnecessary.[1]

Brucia.—This alkaloid also has an intensely bitter taste. Its physiological action is the same as that of strychnia, but seven to ten times weaker. It is less soluble in ether, but more soluble in water, alcohol, and chloroform, than strychnia. Unlike strychnia, it gives no play of colours with nascent oxygen. The special colour test for brucia, a test to which strychnia does not respond, is as follows:—Add a little strong nitric acid, a bright red colour is produced, which, on warming, becomes yellow. If, after warming, a trace of stannous chloride be added, the yellow changes to purple destroyed by excess of stannous chloride, or of nitric acid.

From organic mixtures, strychnia and brucia may be separated by Stas' process, using chloroform, or a mixture of ether and chloroform, as a solvent. If both are present, the alkaloidal residue will respond both to the nitric acid test and the nascent oxygen test. The discovery of both, in a case of poisoning, indicates that probably portions of a plant containing the alkaloids, *e.g.*, nux vomica, has been employed.

Failure to detect Strychnia.—Strychnia is an extremely stable substance, not likely to be destroyed by putrefaction. Richter[2] found it in putrid tissues after eleven years' exposure to decomposition in open vessels. Elimination also of strychnia from the body is comparatively slow. Hence, given that the analysis has been properly conducted, and that a sufficient quantity of material has been submitted to analysis, failure to detect strychnia in the body is strong evidence against death from strychnia poisoning. It is not, however, absolutely conclusive evidence against this supposition, as it is just possible that if death has occurred from a minimum dose of strychnia, and the patient has lived for some time after its administration, complete elimination of the poison may take place before death.

Stimulation of the Spinal Cord is caused also by **Thebaia,** one of the opium alkaloids: this, however, is less active even

[1] Blyth on Poisons, p. 323. [2] *Ibid.*, p. 321.

than brucia. It is also caused, according to Lauder Brunton, by **Calabarine**, one of the alkaloids of Calabar bean. Spasms, more or less tetanic in character, pointing to a stimulant action of the cord, have been observed in poisoning by **Nerium odorum.** Stimulation of the spinal cord resulting in the production of epileptiform convulsions, is one of the effects of **picrotoxin,** the active principle of cocculus indicus; and Blyth mentions as similar in its action to picrotoxin, the poison of **Illicium religiosum,** a plant growing in Japan. **Venous blood,** it may be here noted, irritates the nerve-centres; hence, in narcotic poisoning, and poisoning by drugs tending to cause death by asphyxia, convulsions may precede death, even when the poison itself has no irritant action on the nerve-centres.

Calabar Bean.

The seeds of Physostigma venenosum, N. O. Leguminosæ, a native of Western Africa, Physostigmatis Semina, Calabar beans, are highly poisonous. A dose of twelve grains of the seeds taken for purposes of experiment, caused alarming symptoms in an adult. In 1864, a number of children were accidentally poisoned at Liverpool, by eating the beans; one who had eaten six beans died; and two who had eaten the broken fragments of the kernel of one bean, suffered severely, but recovered. Calabar bean **paralyses the spinal cord,** slows the action of the heart, and causes death by paralysis of respiration. In experiments on animals, large doses have been found to at once arrest the heart's action, and cause death by syncope. In poisoning by Calabar bean, the prominent symptoms are gastric irritation, slight tremors followed by great weakness of the muscles, and slowness of the pulse and of respiration. The mental faculties are unaffected. The pupils are contracted, but the poison appears to act more powerfully on the pupils when locally applied, than when swallowed.

Three alkaloids have been described as present in Calabar bean, viz., physostigmine, eserine, and calabarine. Of these, the first two are considered by many to be identical with one another, and are apparently the constituents to which the paralysing action of the poison is due. Calabarine, according to Lauder Brunton, causes convulsions like strychnia.

Treatment.—General, as for spinal poisons (p. 274). Both atropia and chloral, to a certain extent, antagonise the action of Calabar bean, and have been recommended as physiological antidotes. The antagonism is in neither case complete, but appears to be greater in extent with chloral than with atropia.

Identification.—The beans are kidney-shaped, chocolate coloured externally; and have a broad black furrow, with raised edges lighter in colour than the rest of the surface, running

along the convex border. Dimensions about 1 to $1\frac{1}{4}$ by $\frac{3}{4}$ by $\frac{1}{2}$ an inch; weight about sixty-seven grains. Physostigmine may be extracted from organic mixtures by Stas' process, using benzene as the solvent, and identified by its action on the pupil, and by the red colour given by its sulphate with bromine water.

Gelsemium.

Gelsemium nitidum (*syn.* G. sempervirens), or yellow jasmine, N. O. Loganiaceæ. The dried rhizome and rootlets of this, officinal B.P. (1885), are used in medicine.[1] In overdoses, the drug paralyses the cord, and causes death by paralysis of respiration. Unlike Calabar bean, gelsemium has no very marked action on the heart. Its active properties appear to be due to the alkaloid gelsemine. Wormley estimates that not more than one-sixth of a grain of gelsemine, was contained in a dose of the drug which proved fatal to an adult female. Five fatal cases of poisoning by gelsemine have been reported.[2] The prominent symptoms are muscular weakness, followed by giddiness, frontal headache, double vision, and squinting. The weakness deepens into paralysis, the eyelids drop, vision becomes indistinct, and the muscular power of speech is lost. Respiration becomes slow, and the surface cold. The mind remains unaffected. The pupil is usually contracted, but Ringer points out that gelsemine, when locally applied, causes dilatation of the pupil, or a reverse effect to that produced by internal administration of the drug.

Detection.—Gelsemine may be extracted from organic mixtures by Stas' process, using chloroform as the solvent. The colour tests for it are: (1) with sulphuric acid and manganese dioxide it gives a damask red colour, changing to a rich green, most marked at the edges; and (2) nitric acid strikes with it a brownish green, quickly changing to deep green.

Paralysis of the cord is also the special action of methylconia, a liquid volatile alkaloid allied to conia (see Conium).

[1] According to the B.P. (1885), the medicinal dose of the dried root is five to thirty grains, and of the tincture—strength one to eight—five to twenty minims.
[2] Wharton and Stille (1884), vol. iii., p. 416.

CHAPTER XXIII.

SPINAL POISONS (*continued*).

GROUP 2.—PERIPHERAL POISONS.

Conium.

Conium maculatum, Common or Spotted Hemlock, N. O. Umbelliferæ; Showkran (*Arab.*), Kirdamana (*Bomb.*).—This is a common English plant, growing also in Europe and temperate Asia, the leaves and fruit of which are officinal B.P. and I.P. Cases of poisoning by conium are somewhat rare. Falck[1] found seventeen recorded in medical literature (up to 1880), of which fourteen were accidental, chiefly from the plant being mistaken for parsley or some other harmless herb. One case is recorded of a child, who died poisoned by conium, from blowing whistles made of conium twigs. The fruits are obtainable in the Indian bazaars; but I have not met with any reported Indian cases of conium poisoning.

Conium contains a poisonous liquid volatile alkaloid, conia, and a less poisonous crystalline alkaloid, conhydrine. These are similar in action, paralysing first the peripheral extremities of the motor nerves, and subsequently their trunks. In addition, commercial conia has been found to contain a variable quantity of methyl-conia, a liquid volatile alkaloid, which paralyses the cord. The prominent symptoms of conium poisoning are muscular weakness with loss of power to swallow, the weakness deepening into complete paralysis, affecting the extremities first, and afterwards the trunk. The pupils are dilated, there is ptosis, and sometimes convulsive twitchings. Consciousness remains until asphyxia sets in. Death occurs rapidly, by paralysis of respiration, usually in one to four hours. The medicinal dose of the powdered leaves is two to eight grains, and of the tincture of the fruit—strength 1 to 8—20 to 60 minims. According to Woodman and Tidy, one drop of the alkaloid conia may be regarded as a poisonous dose.

Treatment.—General, as for spinal Poisons.

Post-mortem appearances.—Those of death by apnœa.

Identification.—The stem of the plant is described by Guy

[1] Blyth on Poisons, p. 253.

as tall, smooth, glossy green, and dotted with brownish purple spots. The root is tapering, and in shape something like a parsnip, for which it has been mistaken. The leaves are deep green, and have often been mistaken for parsley leaves, from which, however, they differ greatly in shape.

According to the Pharmacographia, the fruit, as met with in the shops, "consists of the separated mericarps, which are about one-eighth of an inch long. The dorsal surface of these has five prominent longitudinal ridges, the edges of which are marked with little protuberances giving them a jagged or crenate outline. . . . The furrows are glabrous, but slightly wrinkled longitudinally; they are devoid of vittæ." The absence of vittæ distinguishes hemlock fruits from other fruits of the same N.O. All parts of the plant, when bruised and moistened with potassic hydrate solution, give out a peculiar mousey odour.

Conia may be separated from organic mixtures by Stas' process, using petroleum ether as a solvent, and conducting all evaporations, &c., at a low temperature. It is recognised by its peculiar mousey-like odour and its action on animals. Schwarzenbach[1] gives the following colour test for conia: "If dropped into a solution of alloxan, the latter is coloured after a few minutes, an intense purple red, and white needle-shaped crystals are separated, which dissolve in cold potash-lye into a beautiful purple blue." Conia coagulates albumen, and gives an amorphous precipitate with mercuric chloride solution: these characters distinguish it chemically from nicotine (see p. 292).

Other poisonous Umbelliferæ.

Cicuta virosa, water hemlock or cowbane.—This, in Europe, where it is indigenous, has given rise to several cases of accidental poisoning, mostly from the roots being mistaken for parsnips. The prominent symptoms of poisoning by water hemlock are, burning pain in the stomach, nausea and vomiting, giddiness, difficult respiration, dilated pupils, and tetanic convulsions, followed by coma or delirium. Death occurs rapidly. An active principle, to which the name cicutoxin has been given, has been separated from this plant, but its precise chemical nature is unknown. Very similar cases have occurred from the **Œnanthe crocata,** hemlock water dropwort, five-finger root, or dead tongue, and from the **Phellandrium aquaticum,** or five-leaved water-hemlock. This last, however, appears to be less actively poisonous than the others. The **Æthusa Cynapium,** or fool's parsley, is also said to have given rise to similar cases, but Harley has shown it to be non-poisonous.

Curara, Wourali, Urari, or Ticunas.

This is a black resinoid mass, almost wholly soluble in water, used by the South American Indians as an arrow poison. It is believed to be an extract from a species of strychnos, probably S. toxifera, mixed with other matters. When swallowed, it usually

[1] Blyth on Poisons, p. 251.

causes no symptoms of poisoning. Introduced into a wound, it acts like conia, paralysing the motor nerves, and causing death by paralysis of respiration. It contains an alkaloid curarine, sparingly soluble in chloroform, and giving a purple colour with strong nitric acid.

The following **alkaloids** are **similar** in **action** to conia and curara:—**Sparteine**, a liquid volatile alkaloid, contained in common broom (see p. 188). **Staphisagrine**, one of the alkaloids contained in stavesacre (see p. 188), and **Methyl-strychnia**, **Methyl-brucia**, and **Methyl-thebaia**, alkaloids obtained from respectively strychnia, brucia, and thebaia, by the substitution of methyl for hydrogen. It may be noted that this substitution, in the case of the alkaloids just mentioned, converts central into peripheral spinal poisons. In the case of conia, a similar substitution converts a peripheral into a central spinal poison (see methyl-conia, p. 283).

Aconite.

Several of the aconites, N. O. Ranunculaceæ, are highly poisonous, their activity being mainly due to the presence of one or other of two alkaloids, viz., aconitia, and pseudo-aconitia or nepaline. These two alkaloids differ somewhat from one another in composition and chemical properties, but have the same physiological action. Of the poisonous aconites, the most important are—

Aconitum Napellus, monk's-hood, wolf's-bane, or blue-rocket, a perennial plant, two to four feet in height, with dark-green leaves of peculiar shape, and a terminal spike of dark bluish-purple, helmet-shaped flowers; common in England, and growing also in the Himalayas. The root and leaves of this are officinal B.P. and I.P.; and—

Aconitum ferox (see Fig. 35), very similar in appearance to the last,[1] a native of the Himalayas, the root of which forms the greater part of the aconite root of the Indian bazaars, and is stated to be more actively poisonous, even than that of A. Napellus.

Of these, A. Napellus owes its activity mainly to **aconitia,** and **A. ferox** to **pseudo-aconitia**; both contain, also, other less active alkaloids, two of which, viz. aconine and pseudaconine, are obtainable by the action of heat, on respectively aconitia and pseudo-aconitia.

Commercial aconitine varies in composition, and is a mixture

[1] The shape of the leaves of A. Napellus is thus described by Bentley and Trimen:—"Very deeply cut palmately into five or three segments, which are again deeply and irregularly divided into oblong acute laciniæ." Those of A. ferox are less divided, and are described by the same authors as "deeply and palmately cut into five acute irregularly indented lobes."

of aconitia or pseudo-aconitia, with the other alkaloids referred to above. English aconitine owes its activity mainly to pseudo-aconitia, while German aconitine owes its activity chiefly to aconitia. The proportion of aconitia or pseudo-aconitia present, in different specimens of commercial aconitine, varies greatly, and hence different specimens vary greatly in strength.

The aconite root of the Indian bazaars is mainly derived

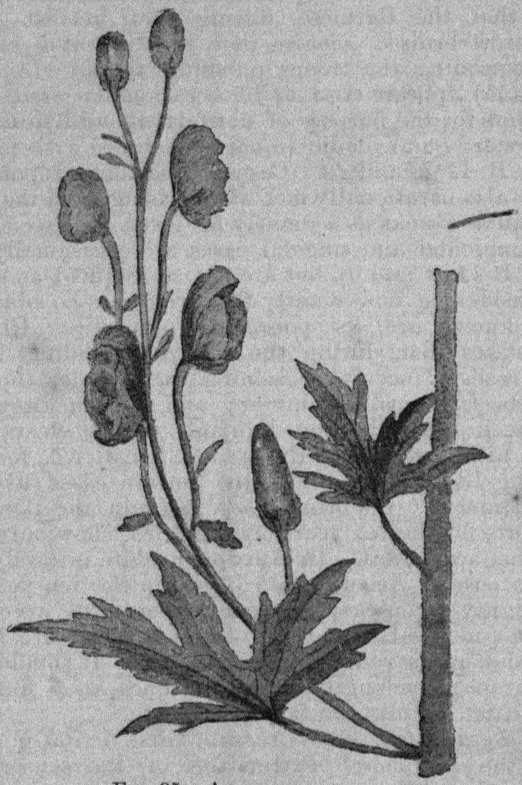

FIG. 35.—ACONITUM FEROX.

from A. ferox, and is known under the names of Bish, Bikh, or Bachnag (*Hind.*), Bachnab (*Bomb.*), Vashanavi (*Tam.*). Other vernacular names for aconite root are Mitha-bish, Mitha-tiliya, Srinji-bish, and Dagra.

Some of the aconites are non-poisonous: *e.g.*, Aconitum heterophyllum, Atis (*Hind.*), Ativish (*Mar.*), Ativadyam (*Tam.*), Ataicha (*Sans.*). The tubers of this are officinal in the Indian Pharmacopœia, and are used in medicine as a tonic and anti-

periodic. They contain an alkaloid called atisine. Other tubers similarly used, and containing the same alkaloid, atisine, are sold under the names of Bikhma, Bishma, or Wakhma. The species of aconite from which these tubers are derived has not been with certainty determined.

In India, in the mountainous tracts of Tirhoot, and in the hill districts generally, a paste made from aconite root is used by native hunters as an **arrow poison.** Chevers, quoting Wallich, mentions that the Burmese, during their retreat before the British, threw bruised aconite root into a water tank in the hope of poisoning the troops pursuing them.[1] Aconite root (like dhatura) appears also to be occasionally used by native liquor dealers for the purpose of **conferring additional intoxicating power**[2] on alcoholic liquor, sometimes with fatal results (see *Cases* B 44 *a* and *b*). Cases of accidental poisoning by aconite are also occasionally met with, arising from the use of the drug by native quacks as a remedy for fever, &c. (see *Cases* B 44, *c* to *e*). Homicidal and suicidal cases are occasionally reported (see *Cases* B 44, *f* and *g*), but are not so frequent as one might expect, considering how readily the drug can be obtained, and how well known are its poisonous properties. Chevers, for example, states that, during the ten years ending 1869, only thirty-six cases of aconite poisoning came under the notice of the Calcutta Chemical Examiner; and Burton Brown records only nineteen cases in the Punjaub in the years 1861–73. Harvey, in his Medico-legal Report for Bengal, &c., for the three years ending 1872, records only ten certain cases (five of them homicidal), and five doubtful cases. Again, the Bombay Analyser's reports for the ten years ending 1884 show only six cases, three of them accidental. **In Europe,** aconite is rarely used for criminal purposes. According to Blyth, in the ten years ending 1882–83, eighty-seven cases of aconite poisoning were recorded in European medical literature, of which two were homicidal, seven suicidal, and seventy-seven accidental. It should be noted that in Europe, accidental cases sometimes arise from aconite root being eaten by mistake for horse-radish.

Action, Symptoms, &c.—Aconite first irritates and then paralyses, the peripheral extremities of the sensory nerves, causing tingling, followed by numbness; first, of the parts with which the poison has been in contact, *e.g.*, the lips and tongue, and subsequently in all parts of the body. This tingling, followed by numbness, is a characteristic symptom of aconite poisoning. Irritation of the stomach is also caused: hence

[1] Med. Jurisp., p. 136.

[2] It may be mentioned here that a non-poisonous bark, viz., that of Acacia leucophlæa, Hewur (*Mar.*), is used in the South Concan in distilling liquor. It contains much tannin, and precipitates albuminous matters present in the juices from which the liquor is distilled: hence its employment.

vomiting, sometimes violent, is generally present. There may be diarrhœa. Besides its action on the sensory nerves, aconite exerts a paralysing action on the motor nerves (or centres), and on the heart. Hence other symptoms of aconite poisoning are, great muscular weakness; the patient, if he attempts to walk, staggers; the respiration becomes slow and weak, and the pulse slow, weak, and irregular. Death may occur from shock or syncope, but usually occurs from asphyxia due to paralysis of the respiration. Convulsions may precede death. The pupil in the early stage of the case, alternately contracts and dilates, but becomes widely dilated in the later stage. The mind is usually quite unaffected, but in exceptional cases delirium has been observed. Case B 45 well illustrates the symptoms of aconite poisoning.

Fatal period.—Shortest recorded, twenty minutes; longest, twenty hours; usual, within three or four hours.

Fatal dose.—One-fiftieth to one-fortieth of a grain of **aconitine** has caused alarming symptoms. Probably one-twentieth to one-sixteenth of a grain given by the mouth would usually cause death in an adult. Blyth considers that the minimum fatal dose, when given by the mouth, is even less than this, and that probably about one-fortieth of a grain subcutaneously injected would cause death. The only officinal preparation of aconitine is an ointment, strength eight grains to the ounce. As regards **the root**, one drachm (presumably of the root of A. Napellus) has caused death. Chevers,[1] however, mentions a case in which fifteen grains of Indian aconite root gave rise to severe symptoms, and bad symptoms have been produced by inhaling the dust arising whilst powdering the root.[2] **The tincture of aconite**, B.P. and I.P., is prepared from the root of A. Napellus, strength two and a half ounces to one pint. The medicinal dose of it is five to fifteen minims. Taylor[3] mentions two cases in which one drachm of the tincture caused death, and a case is reported in which fifteen minims of the tincture caused severe symptoms. In these three cases, the tincture was probably that of the old London Pharmacopœia, which was three times as strong as that of the B.P. Another tincture of aconite, known as **Fleming's tincture**, is three to four times as strong as the B.P. tincture.[4] A case is reported in which death is believed to have occurred from the too frequent external application of **neuraline**, a preparation containing Fleming's tincture. Another officinal preparation of the root, is the **liniment**—strength I.P. one to one,—B.P. two to three. Lastly the B.P. and I.P. both contain an **alcoholic extract** of the leaves (of A. Napellus), medicinal dose one-sixth of a grain gradually increased. Two grains of the extract has caused death.

[1] Med. Jurisp., p. 143.
[2] Woodman and Tidy, p. 394.
[3] On Poisons, p. 756.
[4] Blyth on Poisons, p. 334.

Treatment.—Procure evacuation of the contents of the stomach, administer animal charcoal and stimulants, and keep the patient in a recumbent posture. Apply friction to the surface, and keep up artificial respiration. Blyth recommends hypodermic injection of atropine (four drops of the B.P. solution repeated from time to time); and also if there is great tendency to heart-syncope, the administration of tincture of

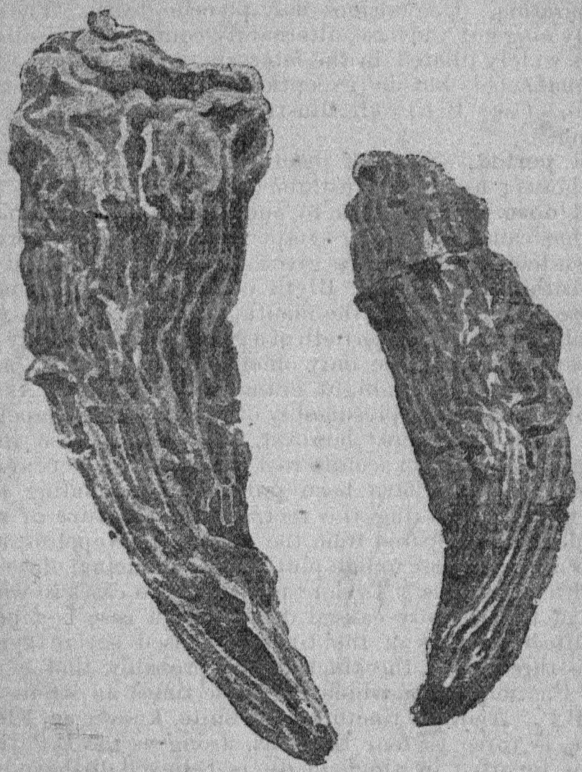

FIG. 36.—ACONITE ROOT (*Aconitum ferox*).

digitalis in half-drachm doses by the mouth, or ten-drop doses subcutaneously. For a case in which the digitalis treatment proved successful, see *Case* B 46.

Post-mortem appearances.—General venous congestion, congestion of the brain and its membranes, and frequently, if the poison has been taken by the mouth, some signs of gastro-intestinal irritation.

Identification.—The root of A. Napellus is conical, or

carrot-shaped, two to four inches long, by half an inch to an inch thick at the top, dark-brown externally, and whitish within. The fresh root has numerous long thin curling rootlets attached to it. The dry root is shrivelled longitudinally and breaks with a short fracture. Chewed, it causes tingling and numbness of the lips, &c. **Horse-radish root** is cylindrical, longer than aconite root, and externally of a light-buff colour. The rootlets attached to it are straight; chewed, it has a pungent taste, but does not cause tingling and numbness like aconite root. **In the Indian bazaars, aconite root** is met with in two forms: (1) Conical roots, three to four inches long, and half to one and three-quarters inches in greatest diameter, shrivelled longitudinally, and often flattened and arched. Externally they are blackish-brown; internally, from being dried over a fire, they are generally hard, horny, and translucent (2) The same roots, after subjection to some soaking process, generally stated to be soaking in cows' urine. These are black, plump, conical tubers, cylindrical in section, tough and moist, and staining the fingers brown when fresh. When dry, they are hard and brittle, and have a reddish-brown resinous fracture. They have a strong, offensive, castor-like odour. **Atis** tubers are smaller, and **Bikhma** tubers about the same size as those of A. ferox, but branched. Both are externally much lighter in colour than the tubers of A. ferox. Both taste simply bitter, not producing tingling and numbness like A. ferox.

Aconitia (or pseudo-aconitia) **may be extracted from organic mixtures** by Stas' process, conducting the evaporations at as low a temperature as possible, and using a mixture of chloroform and ether as a solvent. There are no reliable special colour tests for these alkaloids. They may, however, be identified by physiological tests, viz., by the tingling and numbing sensation produced by a cautious application of a solution of the alkaloid to the lip, and by the effects produced by administration of the alkaloid to small animals.

It has been already mentioned that veratria (see p. 186) has a similar action to aconitia on the sensory nerves. In addition, quebracho bark, the bark of Aspidospermo quebracho, N. O. Apocynaceæ, used in medicine to allay dyspnœa in asthma, &c., and containing several alkaloids, among them aspidospermine and quebrachine, has a paralysing action on voluntary movement and respiration, and appears, therefore, to be a spinal poison. Tobacco and lobelia, also, are spinal as well as cardiac poisons (see pp. 291 and 292).

CHAPTER XXIV.

CARDIAC POISONS.

Of the poisons which act more or less directly on the heart, some appear to exert their influence on this organ, mainly through a direct action on its nerve-supply. Several of the poisons which act in this manner have been already described: *e.g.*, under Irritant Poisons, veratria and the veratrums; under Cerebral Poisons, hydrocyanic acid; and under Spinal Poisons, Calabar bean and aconite. Two important poisons of this kind still remain to be considered, viz., tobacco and lobelia. These are spinal as well as cardiac poisons, and cause death by asphyxia due to paralysis of the respiration. The majority of the poisons, however, acting on the heart, and still remaining for description under the head of Cardiac Poisons, appear to act directly on the cardiac muscle. These tend by arresting the heart's action to cause death by syncope. Like spinal poisons, cardiac poisons leave no characteristic post-mortem appearances.

The general treatment indicated in cardiac poisoning is to try to secure: (1) Elimination, by emetics or the stomach-pump: (2) Prevention of action, by giving decoctions containing tannin: (3) Counteraction of effects, by the administration of stimulants, and the employment of physiological antidotal measures, such as keeping the patient in a recumbent position, keeping the surface warm, employing galvanism, and, if required, artificial respiration.

Tobacco.

Nicotiana Tabacum, Tobacco, N. O. Solanaceæ; Tambaku (*Hind.*, *Beng.*, and *Bomb.*), Pugaiilai (*Tam.*).—The dried leaves of this plant are officinal B.P. and I.P., and form the ordinary tobacco used for smoking, &c. They contain a poisonous liquid volatile alkaloid, nicotia or nicotine, and also an unimportant volatile crystalline substance, nicotianin, or tobacco camphor. Cases of poisoning by nicotine are rare; one celebrated case is, however, on record, viz., the case of Count Bocarmé, convicted of poisoning his wife's brother, by forcible administration of nicotine. Cases of poisoning by tobacco, mostly accidental, are more common. Death has resulted from swallowing tobacco, from

administration of a decoction of tobacco as an enema, and from swallowing tobacco juice such as collects in pipes; and bad symptoms have been caused by the application of tobacco leaves to a wound, and even to the sound skin. Death has occurred from excessive smoking; it is doubtful, however, whether tobacco smoke contains nicotine; probably its poisonous effects are due to pyridene bases, developed during the combustion of the tobacco.

Action, Symptoms, &c.—Tobacco first slows and afterwards quickens the pulse, acting on the heart through the vagus, which it first stimulates, and afterwards paralyses. It is also a spinal poison, and causes death by paralysing the respiration. The prominent symptoms of tobacco-poisoning, are giddiness, muscular weakness, faintness, and depression, abdominal pain, vomiting, sometimes purging, difficult respiration, and convulsions. The pulse is at first slowed, afterwards it becomes quick, weak, and irregular. Death usually occurs rapidly. In one case fatal results followed the administration as an enema, of a decoction of half a drachm of the leaves.

Treatment.—Evacuate the contents of the stomach, give tannic acid and stimulants, keep the patient in a recumbent posture, and apply warmth to the surface. Blyth recommends cautious hypodermic injection of strychnia.

Post-mortem appearances.—Not characteristic; there may be congestion of the brain, lungs, and liver. In some cases, inflammation of the stomach and intestines has been found.

Detection.—Portions of tobacco-leaf may be found and recognised by their odour and physical characters. Nicotine may be extracted from organic mixtures by Stas' process as for conium, and recognised by its odour and action on animals. There are no special colour tests for nicotine. Nicotine does not coagulate albumen, and gives a crystalline precipitate with mercuric chloride solution (distinction from conia).

Duboisia hopwoodii, or piturie, a small shrub or tree belonging to the N. O. Solanaceæ, indigenous in Australia, contains a liquid volatile alkaloid called piturie, which resembles nicotine in physiological action.

Lobelia.

Lobelia inflata, Indian tobacco, N. O. Lobeliaceæ.—The "dried flowering herb" of this plant, a native of North America, is officinal B.P. and I.P. It has long been known to contain a poisonous liquid volatile alkaloid, lobelina or lobelin; and lately a solid alkaloid resembling apomorphia in emetic character, has been found to be also present in it. Several fatal cases of poisoning by lobelia, due to the administration of the

drug by quacks, have been reported in England and the United States.

Action, Symptoms, &c.—Similar to those of poisoning by tobacco, except that there is more burning pain in the stomach, &c. As in poisoning by tobacco, death occurs by paralysis of the respiration. Ten to fifteen grains of the powdered leaves or seeds, will act as a strong emetic, and a drachm of the powdered leaves has caused death. The treatment should be the same as in poisoning by tobacco.

Post-mortem appearances.—Inflammation of the stomach and intestines, and congestion of the vessels of the brain.

Identification.—Lobelia is generally met with as a greenish coloured powder of disagreeable odour, and acrid burning taste. The powder consists of the chopped herb. In the powder the seeds may be found; these are ovate-oblong, about one-fiftieth of an inch in length, are light brown in colour, and have a reticulated pitted surface. According to Guy, 3176 of them weigh one grain.

Lobelia nicotianæfolia, *vern.* Deonal, Bokenal, Dhaval.—This lobelia, Dymock states, is found upon the mountain ranges of Ceylon and Southern and Western India. The upper portion of the stem is hollow, and is dried and used as a shepherd's pipe. The plant has recently been examined, and found to contain the same active constituents as L. inflata.

Identification.—Dymock gives the following description of the plant: "The leaves resemble those of the tobacco; they are finely serrated and covered with simple hairs. The lower part of the stem is woody, an inch and a half or more in diameter, and almost solid; the upper portion is a hollow tube ending in a crowded head of flower spikes; the latter are about a foot in length, and when the plant is in fruit, are thickly set with globular capsules about the size of a pea, to which a portion of the dry flower is often adherent; the capsules are two-celled, each cell containing a fleshy placenta. The seeds are numerous and very small (one-fiftieth of an inch in length), oval, flattened, and marked with delicate lines. Several small tubercles surround the site of the placental attachment; their colour is light brown. The whole plant when dry is studded with small spots of resinous exudation, and is hot and acrid to the taste."

Digitalis.

Digitalis purpurea, or Purple Foxglove, N.O. Scrophulariaceæ (see Fig. 37).—This plant is a native of Europe: all parts of it are poisonous. The leaves, probably the most poisonous portion of the plant, are officinal B.P. and I.P. Several active principles have been described as present in digitalis, of which the

most important are digitoxin, digitalin, and digitalein.[1] Of these, the last is the only one soluble to any extent in water. All three are powerful heart poisons. They stimulate the cardiac muscle and prolong the contractions of the heart, subsequently rendering the heart's action irregular, and finally arresting it. Of the three, digitoxin is said to be five to ten times more powerful than either of the others. Blyth estimates that one-sixteenth of a grain of digitoxin would probably prove fatal to an adult. When boiled with dilute acids, digitoxin yields

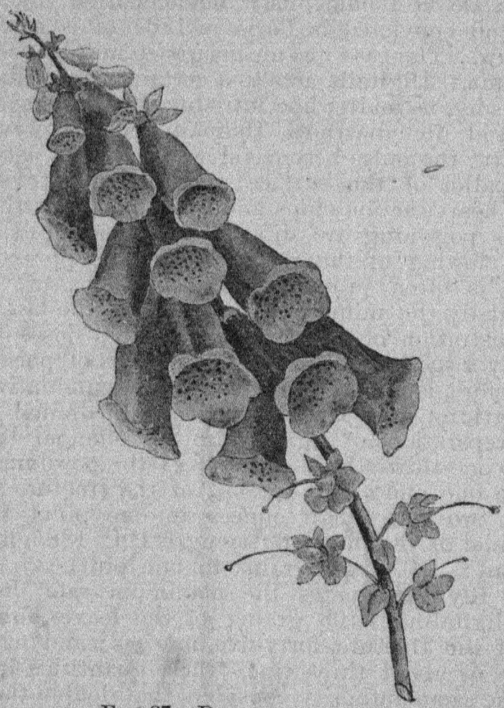

FIG. 37.—DIGITALIS PURPUREA.

toxiresin and digitalin yields digitaliresin; both these also are highly poisonous, but cause convulsions like picrotoxin.

Commercial Digitaline.—Formerly, the active principle of digitalis was stated to be digitaline. This was officinal in the B.P. of 1867, but has been omitted from the B.P. of 1885.

[1] Another principle present in digitalis, viz., digitonin, is readily soluble in water, and appears to have an action like that of saponin. This action is to a certain extent antagonistic to that of digitoxin, digitalin, and digitalein, its tendency being apparently, to depress instead of stimulate the heart muscle.

Digitaline is still officinal I.P. Several varieties of digitaline have been prepared and sold, the chief being:—1. Nativelle's crystallised digitaline, containing digitoxin as its chief constituent. 2. Homolle's amorphous digitaline, containing digitalin as its chief constituent. This is the digitaline of the I.P. and old B.P.; and 3. Soluble digitaline, a large proportion of which consists of digitalein.

Cases of poisoning by digitalis are rare, and are chiefly due to accidents arising out of the medicinal use of the drug. One celebrated homicidal case is, however, on record, viz., the case of Dr. De la Pommerais, a homœopathic practitioner, who was tried and convicted in Paris in 1864, of poisoning a woman named Pauw. The case was an insurance murder.

Symptoms.—Digitalis acts to a certain extent like an irritant poison, causing, no matter how introduced into the system, nausea, vomiting, and often diarrhœa. Its main action, however, is exerted on the heart: the pulse becomes slow, the heart's action irregular, there is pallor of the surface and tendency to syncope, and finally the heart's action stops, and death occurs. Other symptoms of digitalis poisoning are dilatation of the pupils, disturbances of vision, slowing of the respiration, and suppression of urine. Salivation is often present, and convulsions are occasionally seen. Usually the mind remains clear to the last. Sometimes the administration of a series of medicinal doses of digitalis, is followed by a sudden outbreak of symptoms of poisoning. Digitalis, therefore, is generally stated to be a cumulative poison.

Preparations and Dose.—Ordinary medicinal doses of the various preparations of digitalis are—of officinal digitaline, one-sixtieth to one-thirtieth of a grain; of the powdered leaves, half a grain to one and a half grains; of the tincture B.P. and I.P. (strength, two and a half ounces to one pint), ten to thirty minims; and of the infusion (strength B.P. 1885, fifty-six grains to one pint; I.P. sixty grains to one pint), two to four fluid drachms. Blyth estimates the maximum safe dose to be—of officinal digitaline, 0·03 grain; of the leaves, four and a half grains; of the tincture, forty-five minims; and of the infusion, one ounce, or about three times these quantities in twenty-four hours. The same authority considers that double these maximum safe doses would be likely to prove dangerous. A case, however. is recorded of recovery after taking one drachm of the powdered leaves, and another of recovery after swallowing two ounces of the tincture.

Fatal period.—Rarely less than twenty-two hours. In one case death occurred on the sixth day.

Treatment.—Evacuate the contents of the stomach. Give tannic acid and stimulants. Administer aconite cautiously, and keep the patient recumbent.

Post-mortem appearances.—Not characteristic. In some

cases signs of inflammation of the mucous membrane of the stomach and intestines have been present.

Identification.—The flowers are purple and dotted, and bell-shaped. The seeds are minute (about 1126 to the grain: Guy), light brown, angular, and pitted. The leaves are four to twelve inches long, "with a winged petiole of varying length; ovate, or ovate lanceolate, subacute, crenate, or irregularly crenate-dentate, somewhat rugose, slightly hairy, and dull green above, densely pubescent, and paler beneath. Taste very bitter, unpleasant; odour faint, agreeable, and tea-like" (B.P. 1885). Organic mixtures should be treated by the modification of Stas' process, referred to at p. 185, shaking the acid watery filtrate first with petroleum ether to remove impurities, and subsequently successively with benzene, which takes up digitalein, and chloroform, which takes up digitalin and digitoxin. The residues left after evaporation of the benzene and chloroform, must then be tested physiologically. The following colour-test may also be tried, but is not a very delicate one, and cannot be by itself relied on. With sulphuric acid and bromine, digitalin gives a red colour, becoming emerald green on dilution. Digitalein similarly treated, gives a violet colour, becoming light green on dilution.

Nerium odorum.

Nerium odorum, or Sweet Scented Oleander, N. O. Apocynaceæ; Kanir (*Hind.* and *Bomb*), Karabi (*Beng.*), Alari (*Tam.*) (see Fig. 38).—All parts of this common Indian plant are reputed to be poisonous, the root being probably the most active portion. According to H. G. Greenish, Nerium odorum contains two bitter principles, both probably glucosides. To these he has assigned the names respectively of neriodorin and neriodorein. Both are powerful cardiac poisons, appearing to act on the heart in a similar manner to digitalin. Besides acting on the heart, Nerium odorum appears to have an action on the spinal cord somewhat similar to that possessed by strychnia.

In India the poisonous qualities of Nerium odorum appear to be popularly well known. Cases of poisoning by it are, however, not very frequent. Only seven cases were reported to the Bombay Chemical Analyser's office during the ten years ending 1884; and Harvey only records three cases in his Bengal Medico-legal Report for the three years ending 1872. Chevers details seven cases. Of these seventeen cases, nine were suicidal, two homicidal, in two the drug was used as an abortifacient, and in four the drug was given or taken medicinally.

Symptoms.—The administration of this poison is frequently, but apparently not invariably, followed by vomiting, succeeded usually by drowsiness and insensibility. The pulse becomes slow and weak. These symptoms are accompanied by muscular

twitchings deepening into tetanic spasms. Lockjaw is frequently present. The tetanic spasms present in poisoning by Nerium odorum, unlike those present in strychnia poisoning, have been noticed to affect in greatest degree the muscles of one portion of the body. Thus, in one case it is reported that the muscles of the right arm were chiefly affected, and in another, that the spasms, although affecting the whole body, were more developed in the upper than in the lower extremities, and more developed on the left than on the right side (see *Cases* B 47 and 48).

FIG. 38.—NERIUM ODORUM.

Post-mortem appearances.—The mucous membrane of the stomach usually shows signs of irritation. In two out of three cases reported by Chevers, both sides of the heart were found full of dark fluid blood; in the third the right ventricle was distended with dark fluid blood, and apparently the left heart empty.

Treatment.—The general treatment for poisoning by cardiac poisons.

Identification.—A shrub about six or eight feet high, with linear lanceolate leaves and white or red flowers (see Fig. 38).

Dymock describes the root as "crooked, bark thick soft, external surface grey, corky; on young roots the corky layer is very thin, and the interior yellow colour of the bark is seen through it, inner surface yellow. The bark when cut or wounded exudes a pale yellow latex, which is resinous and very sticky. Odour somewhat acrid, like that of a raw potato. Taste acrid and bitter." There are no characteristic chemical tests for the glucosides of Nerium odorum. In cases of poisoning, an attempt may be made, however, to extract them and identify them by their physiological action. Both are soluble in alcohol; neriodorin is sparingly soluble in water, but soluble in chloroform. Neriodorein is insoluble in chloroform and other immiscible solvents, but soluble in water.

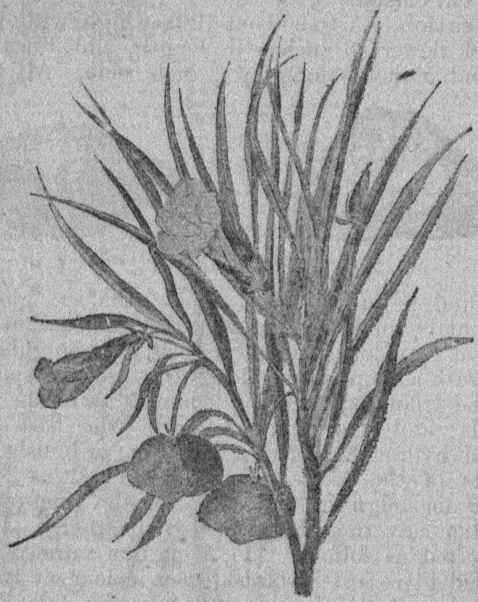

FIG. 39.—CERBERA THEVETIA.

Cerbera Thevetia and Cerbera Odallam.

Cerbera Thevetia, or Thevetia neriifolia, Yellow or Exile Oleander.—Pila-kanír (*Hind.* and *Bomb.*); Kolkaphul and Cheena Kuroopee (*Beng.*); Pach-chai-alari (*Tam.*); N. O. Apocynaceæ (see Fig. 39).—This plant, a native of the West Indies, but domesticated in India, is highly poisonous. It contains a glucoside, thevetin, a powerful heart-poison, acting similarly to digitalin.

In India cases of poisoning by Cerbera Thevetia in the human subject are seldom met with. Of late years, however, the seeds of the plant have come into somewhat extensive use in some parts of the Bombay Presidency as a cattle poison, nine cases of this kind having been reported in the Bombay Chemical Analyser's office during the year 1886.

Symptoms.—The more prominent of these are vomiting and purging, with drowsiness and dilated pupils, and depression of the heart's action. In one case a child, æt. three, died with symptoms of tetanus after eating one seed, and in another case eight to ten of the seeds proved fatal to an adult female. A tincture of the bark has been used in medicine as an antiperiodic; thirty to sixty drops of a tincture, strength one to five, acts as a purgative and emetic.

Identification.—A tree about 12 feet high, with large yellow bell-shaped flowers 3 inches in length, and linear lanceolate leaves about 5 inches long by ¼ inch wide. All parts of the

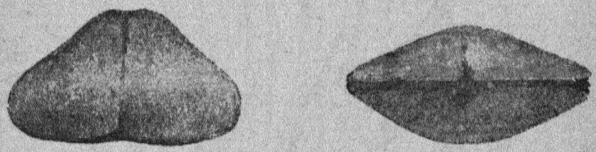

FIG. 40.—CERBERA THEVETIA SEEDS.

plant abound in milky juice. The fruits are globular, light-green, about 1½ to 2 inches in diameter, and contain a single nut, light-brown in colour, and of a peculiar triangular shape (see Fig. 40), with a deep groove along the edge corresponding to the base of the triangle; each nut contains two pale yellow seeds. The seeds and the inner layer of the bark give, when boiled with hydrochloric acid, a deep blue or bluish-green colour. Fragments of the seeds may be recognised as follows:—Exhaust with alcohol, filter, and evaporate the tincture to dryness. The residue may then be washed with ether, and the washed residue tested as follows. (1) A portion warmed with hydrochloric acid gives a deep bluish-green colour, destroyed by permanganate of potash solution. (2) A portion treated with strong sulphuric acid gives a brown colour, changing slowly into a rich crimson, which, on exposure, becomes deep green at the edges.

Cerbera Odallam (see Fig. 41).—This plant, closely allied botanically to the last, probably contains the same poisonous principle. Like the plant last described also, it abounds in milky juice, and this and the seeds when heated with hydrochloric acid give a deep blue or bluish-green colour, similar to that given by Cerbera Thevetia. The flowers are jasmine-shaped, white, about 1 inch in length; the leaves are dark green, fleshy, and lanceolate, about 4 to 5 inches long, by 2 inches in greatest breadth. Fruits

nearly globular, dark green, and contain in a cavity in the centre a single oval white seed.

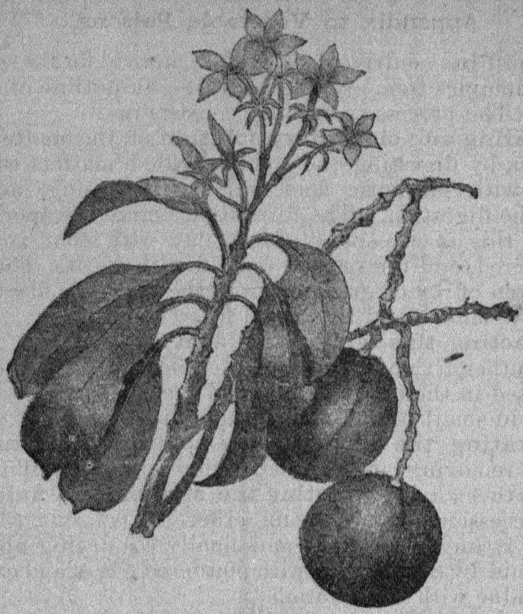

FIG. 41.—CERBERA ODALLAM.

Other Cardiac Poisons.

In addition to the foregoing, the following, already described under Vegetable Irritants, appear to possess an action on the heart similar to that possessed by digitalin. **Scillitin,** the active principle of squill, and probably also superbine, from Gloriosa superba. **Helleborein,** from Helleborus niger and H. viride. **Anemonin,** from Anemone pulsatilla, &c., and **Adonidin,** from Adonis vernalis.

An action on the heart similar to that of digitalin appears also to be possessed by the following:—**Antiarin,** a glucoside contained in Antiaris toxicaria, a native of Java, where the milky juice of the plant is used as an arrow poison. **Strophantin,** a poisonous principle contained in Strophantus hispidus, N. O. Apocynaceæ. **Apocynin,** a poisonous principle contained in the root of Apocynum cannabinum. **Erythrophleine,** an alkaloid obtained from the bark of Erythrophlœum guineense, a native of West Africa. **Euonymin,** a glucoside contained in Euonymus atropurpureus: and by **Tanghinia venenifera,** or Madagascar ordeal poison.

The following, already described under Vegetable Irritants, appear to possess an action on the heart similar to that possessed by digitonin:—**Saponin,** from

soap nuts, &c., **emetine**, and **apomorphine**. A similar action also is possessed by **muscarine**, the alkaloid of Amanita muscaria (see Poisonous Mushrooms, p. 271).

Appendix to Vegetable Poisons.

Dragendorff has contrived a complete method for the separation of plant principles from organic mixtures, an outline of which is as follows. The process essentially consists in—

A. Preparing an acid watery solution of the matters under examination, by digesting the finely divided matters with water acidulated with sulphuric acid, filtering, pressing, and twice repeating the digestion. The filtrates are mixed, evaporated to a syrup, and this is macerated for a day with four volumes of alcohol, filtered, and the residue washed with spirit. The alcohol is then driven off by evaporation from the alcoholic filtrate, water (if necessary) added, and the liquid filtered again.

B. Extracting this while still acid by agitation with (1) petroleum ether, (2) benzene, and (3) chloroform. The solvents are to be used in the order named, and each, after use, separately evaporated in small portions in suitable dishes.

C. Saturating the acid watery solution with ammonia (previously removing any residual chloroform by agitation with petroleum ether), and **extracting the ammoniacal fluid** as in B with, in succession, (1) petroleum ether, (2) benzene, (3) chloroform, and (4) amylic alcohol; and finally (5) drying up the ammoniacal fluid by evaporation with powdered glass, and extracting the dry residue with chloroform.

The following Table shows the principal substances likely to be found in the various residues. Non-poisonous substances are mentioned in brackets thus (Theine).[1]

Solvent.	B Residues from Acid Fluid.	C Residues from Alkaline Fluid.
1. Petroleum ether	Piperine, picric acid, carbolic acid, camphor, and capsicin.	Liquid volatile alkaloids and aniline; also strychnia, brucia, veratria, emetine, and (quinine).
2. Benzene . .	Cantharidin, santonin, digitalin, and (theine); elaterin, colocynthin, colchicin, and absinthin.	Strychnia, atropia, and hyoscyamine; also (quinine, cinchonine, and narcotine), brucia, physostigma, veratria, aconitia, and emetine.
3. Chloroform .	Picrotoxin, helleborin, (cinchonine), digitalein, saponin, and jervine.	Morphia, (cinchonine).
4. Amyl alcohol .	.. ..	Morphia, solanine, saponin, salicin.
5. Chloroform. .	.. .. .	Curarine.

[1] For details of the process, see Blyth on Poisons, pp. 224 *et seq.*

CHAPTER XXV.

SEPTIC POISONS. PTOMAINES.

SNAKE POISONS.

In India a large number of deaths are every year reported as due to snake bite Statistics collected by Fayrer show that in certain Indian provinces 11,326 deaths from this cause were reported during the year 1869, equal to a death-rate of about 93·5 per million of population[1] In the provinces included, the death-rate from snake bite varied from 43 per million (Punjaub), to about 128 per million (Bengal). In one district of Bengal, viz., Burdwan, the snake bite death-rate appears to have been as high as 173 per million.[2] It is quite possible, of course, that some of the deaths reported as due to snake bite are due to some other cause, and the case reported as snake bite, either because the true cause of death is unknown, or with the view of concealing how death occurred. Thus Harvey[3] mentions a case where the body of a man was sent for examination, accompanied by that of a snake which it was alleged had bitten him, and caused his death. On examination the snake proved to be a non-poisonous one, and the cause of death strangulation, the case being one of murder. It is very seldom that death from snake bite is other than accidental. Chevers points out, however, that homicide by snake bite is mentioned in Hindu and Mahomedan law, and that formerly in India criminals were sometimes executed by snake bite. Apparently only one proved case of homicide by snake bite has in recent times been recorded (see *Case* B 49).

Poisonous snakes are distinguished from non-poisonous snakes by possessing poison fangs. These are long, sharp, and curved teeth, either grooved or tubulated, the groove or tubulure communicating by a duct with the poison gland, situated behind the eye. On each side there is only one active fang, but a number of reserve rudimentary fangs lie near it, enclosed in a fold of mucous membrane. The active fangs are firmly fixed to

[1] 'Thanatophidia of India.' The provinces were Bengal, Assam, Orissa, N.W. Provinces, Punjaub, Oude, Central Provinces, and Burma.

[2] Average for ten years ending 1869.

[3] Beng. Med. Leg. Rep., 1870–72, p 159.

the maxillary bones, which, with the exception of the pre-maxillary, are the most anterior of the bones forming the upper jaw. When the active fang is lost, one of the reserve fangs takes its place and functions. In poisonous snakes the maxillary bones are short, and carry either the poison fang (and reserve fangs) only, as in the viperine snakes; or, as in the poisonous colubrine snakes, other teeth as well, posterior to the poison fang, always, however, few in number. **In non-poisonous snakes** the maxillary bones are long, and carry numerous teeth, as a rule all of equal size, but in a few the most anterior of the maxillary teeth are larger than the rest, and resemble poison fangs, but do not, of course, communicate with a poison gland. In addition to the teeth carried by the maxillary bone, both poisonous and non-poisonous snakes have a row of teeth on each side of the middle line of the palate, posterior and internal to the maxillary bones. Hence the upper jaw of the non-poisonous snake has on each side two rows of numerous teeth, while in poisonous snakes the outer row is represented by the poison fang only (viperine snakes), or by the poison fang and one or two other teeth (poisonous colubrine snakes).

Two classes of poisonous snakes exist, viz., 1, **Poisonous colubrine snakes**, and 2, **Viperine snakes**. The main points of distinction between these are shown below.

1. Poisonous colubrine snakes.	2. Viperine snakes.
Head small, and covered by large non-imbricated scales or shields.	**Head** broad and triangular, and, as a rule, covered with small scales.
Maxillary bone carries other teeth, few in number, as well as the poison fang.	**Maxillary bone** carries the poison fang only.
Body, as a rule, long.	**Body**, as a rule, short.

The poisonous colubrine snakes are divided into **hydrophidæ**, or sea snakes,[1] known by their flattened-out tail, and **elapidæ**, or land snakes. These last are again subdivided into **najidæ**, or snakes with hoods, and **elapidæ**, or snakes without hoods.

The viperine snakes are divided into **viperidæ**, or vipers, and **crotalidæ**, or pit-vipers, so called from their having a deep pit on either side of the head, between the eye and the nostril.

Several members of the family Hydrophidæ are found on the Indian coasts; all are very poisonous.

The Indian terrestrial snakes include—

1. **Najidæ**, of which there are two genera, each represented

[1] Fresh-water snakes are not poisonous.

by a single species, viz.:—(*a*) **Naja tripudians**, or cobra, common all over India; and (*b*) **Ophiophagus elaps**, Hamadryad, King cobra, or Sunkerchoor, resembling the cobra, but larger, widely distributed like the cobra, but less common.

2. **Elapidæ**, of which there are three Indian genera, viz.:—(*a*) **Bungarus**, of which two species are common in India, viz., **Bungarus cæruleus**, or Krait, a snake of medium size, white below; bluish black, or brownish black, with narrow white transverse streaks above; probably, according to Fayrer, next to the cobra, the snake most destructive to human life in India: and **Bungarus fasciatus**, or Sankni, larger than the last, and known by its peculiar coloration, consisting of alternate transverse bands of black and yellow surrounding the body. (*b*) **Xenurelaps**, of which only one species is known, viz., Xenurelaps bungaroides, apparently somewhat rare; and (*c*) **Callophis**, a genus of colubrine snakes of small size, with short fangs, of which several species exist in India.

3. **Viperidæ**, of which there are two Indian species, viz.:—(*a*) **Daboia Russellii**, or D. elegans, the Chain viper, Bora, Gunus or Tic polonga; a large very deadly viper of a light-brown colour, having along the back three rows of large, black, white-edged, ovate or circular rings; common in Bengal, and found also in many other parts of India: and (*b*) **Echis carinata**, Afæ, Phoorsa, or Kupper, smaller than the last, brown or brownish-grey in colour; marked along the back, with a row of white brown-edged marks, with whitish curved bands on either side, and on the belly with numerous small dark spots; found in the Bombay Presidency, Sind, and other parts of India.

4. **Crotalidæ**, of which there are four Indian genera, viz.:—(*a*) **Trimeresurus**, of which several species are found in India; many of the most common species are green in colour, and live among the leaves and branches of trees, *e.g.*, the Trimeresurus anamallensis, common on the Ghauts, near Bombay. (*b*) **Peltopelor**, of which one species only is known, viz., Peltopelor macrolepis, a green arboreal crotalus, common in some hill districts. (*c*) **Halys**, of which two species are found in some of the hill districts, viz., Halys elliotti, green above and white beneath, and Halys himalayanus, dark brown or greenish-brown; and (*d*) **Hypnale**, of which one species only is known, Hypnale nepa, or Carawilla, a small crotalus found in Southern India; brown, grey, or reddish olive in colour, with a double row of dark spots on the back, sometimes joining across the back and forming transverse bands.

It may be mentioned that the very poisonous American rattlesnakes, not found in India, are crotalidæ. Of the more commonly met with Indian terrestrial snakes, the most poisonous are the najidæ and daboia, next to these comes the krait; less poisonous than these are the sankni and phoorsa, and the least poisonous of all are the callophides and crotalidæ.

Action and symptoms.—Snake venom has both a **local** and a **remote** action. **Locally it acts as an irritant,** and hence, when introduced into a wound, causes immediate burning pain in the wounded part, followed by swelling and inflammation. Even when applied to thin unabraded membranes such as the conjunctiva, it acts as a local irritant. **Its remote action** is exerted either on the **nervous system,** or on the **blood,** or on both; and may not only be the result of its absorption into the system from a wound, but may even result from its absorption through unabraded delicate membranes, such as the mucous membrane of the stomach.[1] The remote action of the poison of the cobra and other colubrine snakes, seems usually to be mainly exerted on the nervous system. On the other hand, marked symptoms of blood-poisoning, preceded or accompanied by nervous symptoms, great or slight in severity, are a usual result of the bite of the daboia and echis, and probably of other viperine snakes.

Action on the nervous system.—An interval varying in duration usually elapses between the bite of a poisonous snake, and first appearance of nervous symptoms. In the human subject, according to Wall,[2] this interval in cases of cobra bite is usually about an hour, but may be longer; it is often longer in cases of bite by the less venomous snakes. **In cobra bite** in the human subject, the chief nervous symptoms are a feeling of intoxication, followed by loss of power in the legs, the patient staggering or falling if he attempts to walk or stand. The loss of power then spreads to other muscles, those of the tongue and larynx becoming early affected, and the powers of speech and deglutition are lost. About the same time profuse salivation sets in, the saliva trickling away, the power of expelling it having ceased. The paralysis then becomes general, the respiration slow, and consciousness may be lost. Ultimately death occurs by asphyxia, due to gradual stoppage of the respiratory movements. Nausea and vomiting are often early symptoms, and asphyxial convulsions may precede death. The pupil, as a rule, is but little affected. Very similar nervous symptoms usually follow the bite of other colubrine snakes. **Daboia bite** also causes marked nervous symptoms, but the paralysis is more general, does not specially affect the tongue and larynx, and salivation is, as a rule, absent. Convulsions also are often present early in the case, and the pupil is usually dilated. In echis bite the nervous symptoms are as a rule comparatively slight in severity. **Mental shock,** it may be further remarked, may to some extent modify the nervous symptoms present in a case of snake bite; and from recorded cases it appears, that the bite of a non-poisonous snake may give rise to mental shock so severe as to cause death.

Action on the blood.—Symptoms of blood-poisoning may

[1] 'Thalassophidia of India,' p. 64. [2] On 'Indian Snake Poisons,' p. 12.

accompany or follow the nervous symptoms, and appear to be due to the assumption by the blood of a condition of abnormal fluidity, resulting in an excessive tendency to hæmorrhage. Hence discharges of blood take place from the mucous surfaces, the bitten part, abrasions on the skin, &c., and petechiæ may appear from effusion of blood into the subcutaneous cellular tissue. The action of the poison on the blood may modify also the local action of the poison, increasing the severity of the inflammation, and causing sloughing of the wounded part, or even gangrene of a limb. Severe symptoms of blood-poisoning are not usually present in colubrine snake bite, but are a marked feature of daboia and echis bite. They may continue for days after the nervous symptoms have disappeared, may end in death from exhaustion, and in echis poisoning are often the chief symptoms present. Further, owing to this special tendency to blood-poisoning, danger to life in cases of daboia and echis bite may continue long after the nervous symptoms have been recovered from; while in colubrine snake poisoning danger as a rule ceases with the disappearance of the nervous symptoms. To this rule, however, must be excepted the poison of the Bungarus fasciatus. Wall,[1] in experimenting upon animals with the poison of this snake, found that in some cases symptoms were caused by it exactly resembling those seen in cobra bite, while in others the first effects of the poison on the nervous system were slight, and soon passed off, but, after an interval of two to five days, were followed by a fresh set of constitutional symptoms. The animal became weak, purulent discharges took place from the eyes, nose, and rectum, the urine became albuminous, and death occurred from exhaustion several days after the bite. In these cases, however, there was no tendency to hæmorrhage.

Transmission of the poison.—Fayrer[2] mentions a case where an infant died from snake poisoning, the poison having been conveyed through the milk of the mother, who had been bitten and also died. According to the same author, the blood of animals, dead of snake poisoning, if injected into other animals, destroys life. The bodies of animals killed by snake poison may, however, be eaten with impunity by man and animals.[3]

Diagnosis and treatment.—Should the situation of the bite permit, at once apply a ligature above the bitten part. Wall strongly recommends that this should be a thick india-rubber cord or band, wound several times tightly round the limb. Failing this, two or three ligatures, at intervals of a few inches apart, should be, as recommended by Fayrer, tied round the limb, and the one nearest the bite tightened by twisting with a stick. Then using, if necessary, a lens, examine the part supposed to have been bitten. Usually two scratches, short cuts, or punctures,

[1] On 'Indian Snake Poisons,' p. 93. [2] 'Thanatophidia of India,' p. 43.
[3] *Op. cit.*, p. 64.

under one inch apart, will be found. Sometimes the punctures are very minute and barely visible, a drop or two of blood or serum indicating their position. The situation of the poisoned wound having been made out, **free excision** should at once be resorted to.[1] The excision should include not only a portion of the skin about one and a half inches square, but also the underlying tissues for some little depth. As recommended by Wall, the areolar tissue below and around the excised portion of the skin should be dissected away freely, and on parts, into which from their situation the fangs are able to sink in deeply, the excision should be carried still further. Thus, on the fingers or toes, the soft parts should be excised down to the bone, or the finger or toe amputated; and if the bite is on the ball of the thumb, the fascia and a portion of the muscle should be included. After excision the wound should be washed with solution of caustic potash or potassium permanganate, and the ligature removed. The subsequent treatment may consist of the administration of stimulants, the employment of cold affusion, the use of artificial respiration (if there is a tendency to asphyxia), and general treatment of the symptoms as they arise. Main reliance must, however, be placed on early and free excision.[2]

Post-mortem appearances.—The marks of the fangs should be searched for, and if found the state of the tissues around and underlying them noted. Infiltration of blood or bloody serum into these, strongly indicates injection of venom. Internally, the post-mortem appearance most characteristic of death from snake bite, is abnormal fluidity of the blood. In man, from reported cases, this appearance seems to be present both in colubrine and in viperine snake poisoning. In the lower animals it is only found in the latter. The kidneys, lungs, and liver, may be found congested or normal in appearance, and in colubrine snake poisoning particles of food, &c., may be found in the air-passages, having found their way into these, owing to the existence during life, of laryngeal paralysis.

[1] Wall points out that after an incision has been made through the skin, examination of the parts below will reveal to an experienced eye if injection of venom has taken place; and that any signs of irritation indicative of the wound being something more than a mere mechanical puncture, should be taken as showing that the individual has been poisoned as well as bitten.

[2] Several substances, *e g*, potassium permanganate, destroy the activity of snake poison if mixed with it previous to its introduction into the body. These, however, only do so owing to their general action on certain forms of organic matter. Hence once the poison has been introduced into a wound, these substances, owing to their having no special affinity for the poison, are practically useless as remedial agents. The same may be said of all reputed antidotes for the poison which have yet been tried; and also of what are known as snake-stones, *i.e.*, stones which are reputed to have the power of sucking out the poison when applied to the wounded part.

Other Venomous Animals.

Scorpions.—These have in the last joint of the tail a hollow sting, communicating with a poison-secreting apparatus, and serving like the poison fang in snakes, to convey venom into wounds made by it. The local irritant action of the venom is always very severe. Harvey mentions five cases in which children died from scorpion sting in Bengal, during the three years ending 1872; and from cases quoted by Chevers, it would appear that the sting, at any rate of the larger varieties, may cause death in adults.

Centipedes and spiders are also provided with a poison-injecting apparatus connected with their jaws or mandibles. The bites of these cause effects very similar to those produced by the sting of a scorpion. The bite of some varieties of spider appears to cause severe constitutional symptoms, and may even cause death.

Wasps, Bees, and Hornets are all provided with a poison sting. A single sting from one of these is not likely to cause serious effects, except in cases where inflammation and swelling of the part stung interferes with some important function, *e.g.*, respiration. Taylor mentions, however, two cases in which adult females died from shock after, apparently, a single sting of respectively a wasp and a hornet. A number of stings, as in cases where persons are attacked by a swarm of wasps or bees, has frequently caused severe constitutional disturbance, occasionally ending in death.

Contrary to the popular belief, none of the lizards which up to the present have been met with in India, are venomous.

PTOMAINES.

On examination of animal matters other than the viscera of animals killed by poison, alkaloids have been found present therein. These alkaloids, from the fact that their formation is frequently the result of the post-mortem putrefaction of the matters in which they have been found, have been named ptomaines, or cadaveric alkaloids. Apparently, however, similar alkaloids or ptomaines may also be formed in animal tissues during life.

Some of these ptomaines are poisonous, and it has been suggested that some diseases are due to the formation in the body during life of such alkaloids. Very probably also the poisonous properties found sometimes to be possessed by certain articles of food, *e.g.*, milk, cheese, sausages, &c., are in many cases the result of the development of poisonous ptomaines in the food (see asusage poisoning, and poisoning by cheese and milk, *ante*, p. 228).

It is, therefore, quite possible that in the body of an individual,

who has died from some other cause than poisoning, there may on examination be found an alkaloid, which, when tested by administration to an animal, proves to be poisonous.

A test, by which it has been proposed to distinguish ptomaines from vegetable alkaloids, is founded on the fact that ptomaines reduce ferricyanide of potassium to ferrocyanide. Hence, if a solution of ptomaine in weak acid is added to a mixture of ferric chloride and potassium ferricyanide, a blue precipitate appears. It has been shown, however, that alkaloids other than ptomaines, such as strychnia, brucia, and morphia, possess this reducing action. Hence, this test cannot be relied on. Some of these ptomaines have been found to possess a physiological action similar to that possessed by certain vegetable alkaloids, *e.g.*, strychnia and atropia. No ptomaine, however, has as yet been discovered, possessing all the exact chemical characters, of vegetable alkaloids which (like morphia and strychnia) are capable of being identified by distinctive chemical reactions. Hence, the objection that an alkaloid discovered in a case of supposed poisoning, may be a ptomaine produced by decomposition after death, mainly applies to cases in which the vegetable alkaloid supposed to have been discovered, is one which can only be recognised by its physiological action.

CHAPTER XXVI.

QUESTIONS ARISING IN SUITS FOR NULLITY OF MARRIAGE.—IMPOTENCE. VIRGINITY.

According to the law of England, marriage is a contract which may be declared null and void by the Court on proof that either of the parties thereto is incapable of fulfilling its terms, *i.e.*, of consummating the marriage. Hence a suit for the declaration of nullity of marriage may be brought by one of the parties to the contract on the ground that the other is impotent, and incapable of sexual intercourse. To obtain a decree declaring the marriage null and void on this ground it must however be proved: (*a*) **That the incapacity existed at the time of the marriage**; and (*b*) **That it is of such a nature as to be incurable,** or only curable by an operation to which the individual refuses to submit (see *Case* C 1). Obviously, therefore, in suits for the declaration of nullity of marriage, various questions connected with the subject of **impotence or incapacity for sexual intercourse** may arise. These involve the consideration of this subject, and for convenience, with it may be considered the subject of **sterility or incapacity for procreation.**

Again, as a woman seeking a declaration of nullity of marriage may support her statement, that the man with whom she has gone through the marriage ceremony is impotent, by alleging that, although she has cohabited with him, she is "virgo intacta," another question which may arise in such cases is, **Is a certain female "virgo intacta" or not?**

It may here also be mentioned that a marriage may be declared null and void on the ground of the **insanity of one of the parties thereto.** For the questions arising in such a case, see *Insanity*, page 417.

IMPOTENCE AND STERILITY.

Impotence or sterility may exist in either sex, and the existence of one of these conditions does not necessarily imply the existence of the other, *e.g.*, an individual may be sterile, but not impotent; or impotent, but not sterile. Besides nullity of marriage suits, the question of the impotence, or sterility, of an

individual may arise:—(1) **In rape cases.** Here impotence may be pleaded as a defence by the accused (see *Rape*, p. 328), and similarly, in other cases, impotence may be set up as an answer to a charge of adultery. (2) **In cases of disputed right to inherit** (see this subject); where an individual is alleged to be an illegitimate, or a supposititious child. (3) In some cases where, under certain circumstances (see p. 314), a woman seeks to have absolute control given to her over money, on the ground that she has no children, and is past the age of child-bearing. We may now proceed to the consideration of impotence and sterility in each sex.

(A.) Impotence and Sterility in the Male.

A male may be impotent or sterile owing to, 1. Extreme youth. 2. Advanced age. 3. Malformation or defect. 4. Disease. 5. Mental causes.

1. **Extreme youth.**—According to the **law of England,** the earliest age at which a male can contract a valid marriage is fourteen; and a male under the age of fourteen is held incapable of committing a rape. It appears, therefore, to be a presumption of English law, that a boy does not attain puberty, and become potent, until he has reached the age of fourteen. The **law of India** contains no similar presumption; a boy under the age of seven is (Indian Penal Code, sect 82) held to be incapable of committing rape or any other offence. Over that age, the question of his capacity to commit rape, is a question left to the Courts to decide according to the evidence produced in the case.

The actual age at which males attain puberty, and become potent, varies. Probably the general age among Europeans is about fourteen, and among natives of India somewhat earlier. In exceptional cases puberty is attained at a very early age. Tidy mentions a case of a boy who was given to masturbation from the age of three, and of another boy aged four and a half, who attempted intercourse with his sister aged two.[1] In other cases puberty is not attained until a comparatively late age. Taylor mentions a case of a man whose penis and testicles at the age of twenty-six, "but little exceeded in size those of a youth of eight years of age." This individual married, became the father of a family, and at the age of twenty-eight the organs became fully developed.[2]

The attainment of puberty and consequent potency does **not,** however, **necessarily** imply coincident attainment of **fertility.** Until spermatozoa appear in the seminal fluid, an individual is sterile. Casper considers that the power of procreation

[1] Tidy's Legal Medicine, ii., p. 77. [2] Taylor, ii, p. 290.

commences later (and ceases earlier) than the capacity for coitus. Taylor gives fourteen as the earliest age at which the procreative power has been recorded to appear in the male.[1]

2 **Advanced age** may of course be a cause of impotence or sterility in the male. Cases, however, are recorded of the procreation of children by men of seventy-one, eighty-one, and ninety-two; and spermatozoa have in several cases been found in the seminal fluid (indicating fertility) of men over ninety.[2] Casper once found them in a man aged ninety-six.[3]

3. **Malformation or defect.**—Impregnation may result from the mere deposition of semen within the vulva.[4] No malformation or defect of the penis, therefore, can be regarded as an undoubted cause of impotence, unless it is of such a nature as to completely prevent such deposition. This being so, impotence results from **complete loss or absence of the penis**, or from its **orifice being situated**, as in complete hypospadias or epispadias, in such a position, that deposition of semen within the vulva during coitus is impossible. Impotence has thus resulted from **perinæal fistula**. For the reason above mentioned, impotence is not necessarily the result of partial absence or loss of the penis, or of partial hypospadias or epispadias. **Adhesion of the penis** to the scrotum or abdomen, may cause impotence remediable by a slight operation

Individuals impotent from malformation or defect of the penis are **not necessarily sterile**, it being possible to effect impregnation by artificial injection of the seminal fluid.

Loss of both testicles, or absence of both testicles, of course involves impotence and sterility. The power of procreation may, however, remain for a limited period after the removal of both testicles, owing to presence of accumulated seminal fluid in the vesiculæ seminales.

Loss of one testicle only does not result in impotence, nor are those who have one testicle only (monorchids) impotent (see *Case* C 4). Individuals in whom the testicles have not descended (cryptorchids) are not necessarily impotent; many, but not all, are, however, sterile (see *Cases* C 2 and 3).

4. **Disease.**—**Local disease** may cause temporary and remediable impotence; *e.g.*, elephantiasis, from mechanical obstacle to coition; and stricture of the urethra, from mechanical obstruction to the flow of semen. Local disease may also cause permanent and incurable sterility, *e.g.*, advanced disease of the testicles, or wasting of the testicles after inflammation; this last has been observed as a result of metastatic parotitis. **Lithotomy** has been known to cause sterility, probably from injury to the ejaculatory ducts.[5]

[1] Taylor, ii., p. 285.
[2] *Ibid.*, p. 291.
[3] Casper, ii., pp. 258, 291.
[4] Tidy, iii, p. 14.
[5] Teevan, quoted by Woodman and Tidy, p. 685.

Constitutional diseases, if of an exhausting nature, may produce temporary impotence; but general diseases, not affecting the brain or spinal cord, or not producing great debility, do not usually cause impotence.[1] Injury or disease of the brain or spinal cord may cause impotence or sterility. Curling relates several cases of impotence caused by blows on the head, especially on the back and under-part of it; in some cases of this class recovery takes place, but in others wasting of the testicles and permanent sterility follows.[2] **Paraplegia** from injury to or disease of the cord, according to Curling (and probably also locomotor ataxy), has no direct effect on the testicles, but may cause impotence by destroying the power to copulate. Wasting of the testicles and sterility may, however, follow. In one case quoted by Curling, a man suffering from paraplegia of some years' duration, retained sufficient sexual power to have prolific intercourse. **Hemiplegia** may cause impotence, but Guy mentions two cases where men, within three weeks of an attack of hemiplegia, had sexual intercourse with their wives, and begat children[3] (see *Case* C 5). **Over-indulgence in certain intoxicating** or narcotic **drugs,** *e.g.*, alcohol, opium, cannabis and tobacco, is said to cause sterility. Sterility has by some been stated to occur in chronic lead-poisoning.

5. **Mental causes.**—Excess of passion, timidity, fear, &c., may cause temporary impotence. Individuals may, it is alleged, be impotent or sterile as regards a particular female, but not as regards others (see *Cases* C 4, 6 and 7).

(B.) Impotence and Sterility in the Female.

A Female may be impotent or sterile owing to (1) Extreme youth; (2) Advanced age; (3) Malformation or defect; (4) Disease.

1. **Extreme youth.**—According to the **law of England,** twelve is the earliest age at which a female can contract a valid marriage. It would therefore appear that the law of England presumes, that a girl attains puberty and becomes potent at the age of twelve. According to the **law of India** (Penal Code, sect. 375), any female over the age of ten can give a valid consent to sexual intercourse; and "sexual intercourse by a man with his own wife, the wife not being under ten years of age, is not rape." Hence it would appear that the law of India presumes, that a female attains puberty and becomes potent at the age of ten.

The commencement of fertility in the female is as a rule indicated by the commencement of menstruation. In a few cases, however, a periodical discharge of blood from the genitals

[1] Taylor, *l.*, p. 292. [2] *Ibid.*, p. 294. [3] For. Medicine, p. 42.

has been established in female children of three, two, and even one year of age. In other cases pregnancy has occurred before the appearance of menstruation. No case of pregnancy at an earlier age than eight to nine has been recorded. The actual age of commencement of menstruation varies greatly. Among natives of Europe the general age at which it first appears is fourteen to fifteen. Out of 2000 cases, menstruation appeared in 211 between the ages of ten and twelve, in 1462 between thirteen and sixteen, and in 318 between seventeen and twenty. In one case only did it appear as early as nine, and in one only as late as twenty-two.[1] Among natives of warm climates menstruation occurs earlier than among natives of temperate climates. Chevers states that among natives of India menstruation at the age of ten is very uncommon, but that its appearance is seldom delayed beyond the thirteenth year. The menstrual flow commonly lasts three to four and a half days. The menstrual period, reckoned from commencement of flow to commencement of flow, save in exceptional cases, is twenty-eight days.

2. **Advanced age.**—Menstruation usually ceases at forty to fifty, but has been known to cease as early as twenty-three. On the other hand, there are on record several cases of menstruation at over sixty years of age, and one as late as seventy-seven. As a rule fertility ceases with the cessation of menstruation, but Taylor[2] mentions a case of a lady aged forty-four, who was delivered of her tenth child eighteen months after the entire cessation of the menses. No general rule can be laid down as to the age at which, in the female, fertility ceases.

The question whether a woman is past child-bearing is of practical importance in cases where money has been settled on or bequeathed to a woman absolutely in case she has no children, but in case of having children, only for her life with remainder to her children. In these and similar cases, where the woman has attained an advanced age without having had children, it is presumed she is incapable of having issue, and she is held absolutely entitled to the money in which, if she had children, she would only have a life interest. No particular age is fixed as the period when such presumption arises.

The earliest age on which such a presumption has been acted on is that of a woman aged forty-nine years and nine months (see *In re Millner's Estate*, L. R. 14 Eq., p. 245), but in that case she had been married for twenty-six years and there had never been any children. In the case of *In re Widdow's Trusts* (L. R. 11 Eq., p. 408) the presumption was acted on in the case of a widow aged fifty-five years and four months, and a spinster aged fifty-three years and nine months. In *Norton* v. *May* (9 Ch. Div., p. 388) the Court refused to presume no possibility of issue in the case of a woman aged fifty-four years and six months, who, although she had been married several years, had separated

[1] Hogg, in 'Medical Times and Gazette,' 1871, vol. ii., p. 555.
[2] Medical Jurisp., vol. ii., p. 299.

from her husband soon after marriage, and had only lived with him for about three years before the filing of the case in Court. In *Davidson* v. *Kimpton* (18 Ch. Div., p. 213), the presumption was acted on in the case of a spinster in her fifty-fifth year. There is no similar presumption as to a male being incapable of begetting issue.

3. **Malformation or defect.**—Complete absence or occlusion of the vagina of course causes impotence. Occlusion may be remediable by operation. For occlusion to act as a cause of sterility it must be complete; "the slightest aperture will often suffice for impregnation."[1] Many cases are recorded where during labour the vagina has been found occluded to so great an extent as to require incision in order to effect delivery. Ogston mentions a case of "a woman who had a vagina so narrow as scarcely to admit a quill," but who, after being married eleven years, became pregnant, when the vagina "dilated of itself sufficiently to admit of delivery at the full term."[2] Absence of the ovaries or uterus of course causes incurable sterility. In such cases, however, there may be no external defect or malformation, and the cause of the sterility may in consequence only be ascertainable after death.

4. **Disease.**—Impotence or sterility in the female may arise from a variety of diseased conditions. There may, for example, be excessive irritability of the vagina, preventing coitus and causing impotence (see *Cases* C 8 and 9). Again, effective coitus may be prevented by ruptured perinæum, or recto-vaginal fistula. Sterility may result from disease of the ovaries; obstruction of the Fallopian tubes, or of the neck of the uterus; displacements of the uterus, &c., &c. Discharges from the vagina or uterus may cause sterility by acting destructively on the spermatozoa, or preventing their access to the ovum. Paraplegia in the female, it may be noted, does not always prevent either impregnation or delivery.[3]

VIRGINITY.

It has already been pointed out that one of the questions which may arise in nullity of marriage suits is, **Is a certain female "virgo intacta" or not?** The same question may also arise in other cases; *e.g.*, (1) **In divorce cases and defamation cases.** Here evidence that a certain female is "virgo intacta" may be put forward as an answer to an allegation of adultery, relied on as a ground for divorce, or forming the defamatory matter complained of (see *Cases* C 11 and 12). (2) **In cases where an unmarried female is alleged to be a prostitute,** and as such liable to be dealt with under the Contagious Diseases Act or other similar law (see *Case* C.10). (3) **In rape cases.**

[1] Taylor, p. 302. [2] Ogston, p. 85.
[3] Woodman and Tidy, p. 684.

In these, however, it is not an essential question, seeing that vulval penetration is all that is necessary to constitute the offence of rape (see *Rape*, p. 319), and this may be effected without destruction of the signs of virginity. Premising that in many cases a definite answer to the question under consideration cannot be given, we may now proceed to discuss the evidence upon which the answer depends, viz.:—

The Signs of Virginity.

The most reliable sign of virginity being the presence of an intact hymen, we have to consider the following questions:—1. Is the hymen always present? 2. What is the natural condition of the hymen in the virgin? 3. What are the changes produced in the hymen by sexual intercourse? 4. How far can virginity or non-virginity be inferred from the condition of the hymen?

Question 1. Is the hymen always present?—At one time it was alleged by many authorities that the hymen was frequently absent. This view has, however, been proved by later observations to be erroneous. It was found, for example, to be present in each one of 650 cases examined by Devilliers, Orfila, and Tardieu.[1] Capuron, however, records a case of congenital malformation of the genitals with absence of the hymen.[2]

Question 2. What is the natural condition of the hymen in the virgin?—Ogston[3] gives the following description of the natural condition of the hymen: "In children this membrane lies deep, and can only be readily seen in them on the forcible separation of the thighs, when it is found to consist of two lateral and vertical folds of integument at the entrance of the vagina, which it almost closes. A very common form of the membrane, after the age of infancy especially, is that of an irregularly circular diaphragm, broken at its upper third by an opening more or less large, and placed more or less distant from the lower border of the vaginal orifice. In a third set of cases, the hymen has been described in late observations as a sort of diaphragm, exactly and regularly circular, pierced by a central opening. A fourth form assumed by it, and that its most common appearance, is that of a semicircular fold of integument stretched across the lower border of the vaginal orifice, its free border concave and notched (*échancré*), and its extremities losing themselves in the labia minora. Lastly, the hymen has been occasionally encountered in the shape of a more narrow fringe around the entrance to the canal of the vagina; in one case as a sort of bridle across the vagina, with a passage on each side; in another as a complete septum, pierced by numerous minute openings;

[1] Ogston, p. 102. [2] Tidy, ii., p. 97. [3] Ogston, p. 102.

and in a third instance as a double septum, without any opening whatever into the vagina. The entrance to the vagina is thus practically closed or narrowed by the hymen, which in early life is most usually vertical, but by the natural development of the parts gradually assumes a horizontal direction. Towards puberty more firm and consistent than in early life, as menstruation becomes established it becomes more or less flaccid, presenting less resistance to their flow, and is more easily lacerated."

Question 3. What changes are produced in the hymen by sexual intercourse?—As a general rule, when sexual intercourse takes place, the hymen is lacerated or ruptured, in the latter case giving rise to "those small pyramidal tubercles, from three to six in number, known as the carunculæ myrtiformes."[1] Tidy,[2] however, points out that two or more of the carunculæ "are vaginal and not hymeneal," and that "the vaginal carunculæ may therefore co-exist with an unruptured hymen." If, however, the aperture in the hymen be larger than usual, or the membrane itself be lax, repeated intercourse may take place without rupture, or even laceration. Many cases are recorded, in fact, where the hymen has existed all through pregnancy, and has only ruptured at the time of delivery. In very young children the hymen, owing to its deeply seated position, and to the narrowness of the parts,[3] is not usually even lacerated by intercourse.

The hymen may be ruptured by the introduction of foreign bodies other than the penis: *e.g.* (1) accidentally,—this however is extremely rare; or (2) by the introduction of instruments during an examination or surgical operation; or (3) in practising masturbation, especially if the body introduced is of large diameter; or (4) in endeavours to dilate the parts of young females, so as to render them aptæ viris. Casper mentions a case where the mother of a girl aged ten employed first her fingers and then a long stone for this purpose, thereby lacerating the hymen.[4] Chevers mentions the use in India for this purpose of the fruit of the plantain, and also of pieces of sola pith, the girl being made to sit in water, so that the pith may swell and dilate the parts.[5]

It has also been asserted that the hymen may be ruptured by indirect violence in a fall, or during violent exertion; this appears, however, to be very doubtful. Ogston and Tidy both regard rupture of the hymen in this way as very improbable.[6] The same may be said in regard to the possibility of rupture occurring from the action of vaginal discharges.

Question 4. How far can virginity or non-virginity be inferred from the condition of the hymen?—Given that the

[1] Guy, For. Medicine, p. 48.
[2] Legal Medicine, p. 198.
[3] *Ibid.*, p. 201.
[4] Taylor, ii., p. 430.
[5] Chevers, p. 689.
[6] Ogston, p. 103; Tidy, ii., p. 197.

hymen is intact (not even lacerated), the probabilities, except in the case of females below the age of puberty, are very strongly in favour of virginity; and the inference of virginity becomes almost certain, if the membrane is normal in position and structure, and its aperture is of small size and undilatable; and if accompanying this condition of the hymen the other signs of virginity (see below) are present.

On the other hand, the **absence of an intact hymen**, although strong evidence of non-virginity, cannot be taken as conclusive proof thereof, seeing that, as already stated, the hymen may be ruptured or lacerated by the introduction of foreign bodies other than the penis.

Other signs of Virginity.

These may be briefly stated to be (1) the absence of all signs indicative of previous delivery (see *post*, p. 369); (2) a narrow and rugose condition of the vagina, the clitoris unenlarged, and the labia elastic and in close contact; and (3) a plump and elastic condition of the breasts. All such signs, taken by themselves, are unreliable as evidence of virginity, but are useful as corroborating evidence of virginity derived from the condition of the hymen.

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CHAPTER XXVII.

RAPE AND UNNATURAL OFFENCES.

RAPE.

ACCORDING to the law of India,[1] and also according to that of England,[2] rape—subject to certain explanations—may be defined as sexual intercourse by a man with—(1) Any female under the age, in India, of ten; in England, of thirteen: or (2) Any female over the above-stated age, not being the man's own wife—(*a*) against her will, or (*b*) without her consent, or (*c*) with her consent, if this has been obtained in certain ways, and is thereby invalid. The explanations referred to are as follows:—

1. **As regards the meaning of the term "sexual intercourse,"** or to "unlawfully and carnally know," the phrase employed in English law. It has been decided in England, that any introduction of the male organ within the vulva, constitutes penetration sufficient for the carnal knowledge necessary to the offence. In India, the rule on this point is laid down in the explanation attached to sect. 375 of the Penal Code as follows:—"Penetration is sufficient to constitute the sexual intercourse necessary to the offence of rape," and in the case of *Reg.* v. *Ferroll* (Bombay High Court Sessions, February, 1879), Green, J., directed the jury that vulval penetration only was sufficient, under the law of India, to constitute rape (see *Case* C 13).

2. **As to the male accused.**—As already pointed out, it is an irrebuttable presumption of **English law** that a boy under the age of fourteen is incapable of committing rape. In England, therefore, a boy under the age of fourteen cannot be convicted of this offence. The criminal **law of India** contains no special presumption as to the age at which a boy attains potency, and becomes capable of committing rape. It, however, contains two general exceptions (see *ante*, p. 2), bearing on the question of age in regard to criminal responsibility, and applying to rape as to other offences. These exceptions are in effect (1) that a child under the age of seven cannot be held criminally responsible for his acts, and (2) that a child between the ages of seven and twelve

[1] Indian Penal Code, sect. 375.

[2] Criminal Law Amendment Act, 1885 (48 & 49 Vict. ch 69, sect. 4).

can only be held criminally responsible for his acts if he has attained a certain specified degree of maturity of understanding.[1] Hence, in India, if the case of a boy charged with rape does not fall within one or other of these general exceptions, the question of the capacity of the accused to commit the offence is left to the Court to decide according to the evidence produced in the case. Thus, "in the case of *Kureem Noorbae* v. *Meun Noorbae* (2 N. A. Rep., p. 87), a boy of ten years was convicted for rape by the Court of Sessions, but the Nizamut Adawlut, considering it unadvisable to admit his capability, viewed the matter only as an attempt."[2]

3. **As to the age of the female.**—According to the law of India, sexual intercourse with a female, of or over the age of ten, with her valid consent, is not an offence. According to the law of England, however, sexual intercourse with a female of or over the age of thirteen, but under that of sixteen, with her consent, is (unless it be proved that the accused had reasonable cause to believe that the girl was of or above the age of sixteen) a misdemeanour punishable less severely than rape (Criminal Law Amendment Act, 1885, sect. 5).

4. **As to the consent of the female.**—According to the **law of India**, consent of a female to sexual intercourse is not valid, and does not exculpate the accused, if the manner in which it has been obtained falls within (*a*) certain general exceptions of the Penal Code in regard to consent (see sect. 90); or (*b*) certain special exceptions in regard to consent to sexual intercourse embodied in sect. 375 of the Code.

Hence the consent of the female is invalid:—(1) If it is given under misconception of fact, and the man knows, or has reason to believe, it was so given (sect. 90). (2) If the woman, by reason of unsoundness of mind, or intoxication, is unable to understand the nature and consequence of the act to which she consents (*ibid*). (3) If her consent has been obtained by putting her in fear of death or hurt (sect. 375). (4) When the man knows that he is not her husband, and that her consent is given because she believes that he is another man to whom she is, or believes herself to be, lawfully married (sect. 375). It may be remarked, as regards these exceptions—

(1) That, as regards exception 1, a female, for her consent to be valid, must be aware that the act to which she consents is sexual intercourse. On this point the law of England agrees with that of India (see *Case* C 14).

(2) That, as regards exception 2, the law of England appears to differ somewhat from that of India, it having been decided in the case of *R.* v. *Fletcher*,[3] in which a man was charged with rape on an idiot, that "a consent resulting from a mere animal instinct

[1] Indian Penal Code, sects. 82 and 83. [2] O'Kinealy's Penal Code, p. 177.
[3] L. R. 1 C. C. R. 39; Tidy, ii., p. 194.

would suffice to prevent the act from constituting a rape." By sect. 5 of 48 & 49 Vict. c. 69, however, sexual intercourse with "any female idiot, or imbecile woman or girl, under circumstances which do not amount to rape, but which prove that the offender knew, at the time of the commission of the offence, that the woman or girl was an idiot or imbecile," is a misdemeanour punishable less severely than rape.

(3) That the law of England agrees with that of India in regard to exception 3.

(4) That, as regards exception 4, it was ruled in *R.* v. *Barrow* (L. R. 1, C. C. R. 156), that this is not rape by English law. Since then, however, it has been enacted that a man who "induces a married woman to permit him to have connection with her by personating her husband," is guilty of rape (Criminal Law Amendment Act, 48 & 49 Vict. c. 69, s. 4).

Cases are mentioned by foreign writers of females compelling young boys to have intercourse with them, and Chevers (p. 704) mentions cases as having occurred in India, in which there was good reason to suppose that young boys had, under compulsion, intercourse with females. Cases of this description, however, do not come under the definition of rape laid down in the Indian Penal Code. Under the Penal Code of France, it is an offence for a woman to attempt sexual intercourse, with or without consent, with a boy under the age of eleven.

Certain questions which may arise in rape cases may now be considered, viz.:—1. Can a man unaided, commit a rape on an adult female of ordinary strength, in full possession of her senses? 2. Can a female during sleep, be violated without her knowledge? 3. Does pregnancy ever follow rape? 4. May rape cause death? 5. Can menstrual be distinguished from other blood? 6. How may stains of seminal fluid be identified?

Question 1. Can a man unaided commit a rape on an adult female of ordinary strength, in full possession of her senses?—It has been alleged that this is impossible. That, however, in exceptional cases, rape may be committed under the circumstances stated, is shown by *Case* C 15, reported by Casper, who, in regard to it, remarks, "The interest of this important case cannot be mistaken, for it shows that a healthy powerful woman was certainly completely violated by a single man."[1] Ogston, also, in reference to this question remarks, that the arguments advanced against the possibility of intercourse under the circumstances stated, "apply rather to the case of entire penetration of the vulva, than to the partial entry, which is now admitted in law as amounting to the crime of rape. That such entry may be forced in an ordinary case, I had the assurance of actual fact in at least one serious case."[2] Chevers, again, gives two cases in

[1] Casper, iii., p. 311.

[2] Ogston's Lectures, p. 120.

which rape was effected by unaided single men on adult females. Of course, the younger and weaker the female, and the stronger the man, the greater the probability of the commission of the offence being possible; *Case* C 16 illustrates this. A very old woman also may be incapable of offering sufficient resistance. Chevers mentions a case in which a man committed a rape on a woman of seventy.

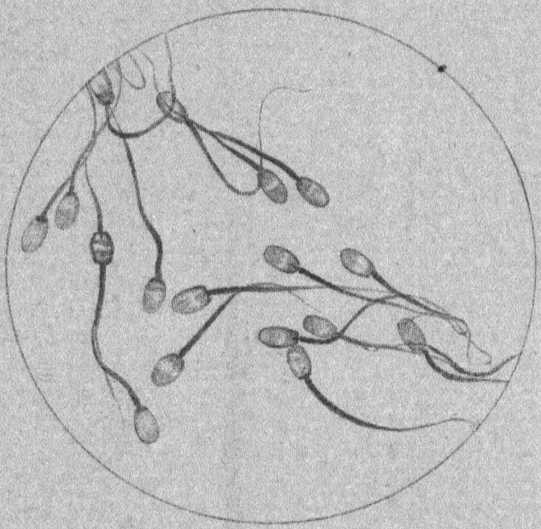
Question 2. Can a female during sleep be violated without her knowledge?—A woman can undoubtedly be violated without her knowledge while under the influence of narcotics (alcohol included), or during syncope or coma; and it has been alleged, also, but this is open to grave doubt, during mesmeric trance (see *Case* C 17, and Taylor's remarks thereon). It is highly probable also that, in exceptional cases, a woman accustomed to sexual intercourse may be violated during profound natural sleep. Guy, in support of this view, mentions the case of a woman, who in illustration of a symptom which somewhat alarmed her—viz., that her sleep was unnaturally heavy—told him that her husband had assured her, that he had frequently had connection with her during sleep.[1] On the other hand, it is highly improbable that a virgin could, during natural sleep, be violated without her knowledge, or even that, without her knowledge, sexual intercourse sufficient to constitute rape could be effected with her. Still, however, cases are reported where it is alleged that this has occurred (see *Cases* C 18 and 19).

Question 3. Does pregnancy ever follow rape?—It was formerly alleged that pregnancy never followed rape, and that hence if a woman charged a man with committing a rape upon her, and became pregnant as a result thereof, the charge must be untrue, and the woman must have consented to the intercourse. Impregnation is, however, independent of volition on the part of the female, and hence pregnancy, as is proved by more than one recorded case, may undoubtedly follow rape.

Question 4. May rape cause death?—The introduction of the male organ into the vagina of an immature female, may produce local injury sufficient to cause death from hæmorrhage, shock, or subsequent inflammation. Harvey[2] records that in Bengal, in the three years ending 1873, 205 cases of rape were proved, of which 133 were on females of or under twelve years of age. In 24 of these laceration of the vagina, generally of the posterior wall, was found; and in 14 the perinæum was torn, the rent varying from one-fifth of an inch, to one inch in length. Five of the cases terminated fatally (see *Case* C 20). Injury to the genitals of a young female may, however, be caused in order to support a false charge (see *Case* C 21). Violent sexual intercourse in a young female at or near the age of puberty, may cause constitutional disturbance, leading to fatal hæmorrhage

[1] For. Medicine, p. 57. [2] Beng. Med. Leg. Rep., 1870–72, p. 179 *et seq.*

XLII.



Page 323. HUMAN SPERMATOZOA × 600.

From Micro-Photograph by Dr. Heneage Gibbes.

Thacker, Spink, & Co., Calcutta.

into the brain, peritoneal cavity, &c.[1] On the question whether death may result from nervous exhaustion, the result of repeated intercourse, Chevers[2] mentions the case of certain Marquesan women, who boasted, apparently with truth, of having had intercourse with one hundred men in one night. The intercourse, however, was voluntary; had it been otherwise, no doubt the exhaustion would have been greater.

Question 5. Can menstrual be distinguished from other blood?—The menstrual fluid is acid, has a sour odour, contains mucous globules and epithelium scales, and less fibrin than ordinary blood. These characters may possibly enable the menstrual fluid seen while flowing, to be distinguished from blood flowing from a wound. Stains, however, of menstrual blood on linen, &c., cannot be positively distinguished from stains of other blood. (See *Detection of blood stains*, p. 73.)

Question 6. How may stains of seminal fluid be identified?—The distinguishing character of the seminal fluid is the presence of **spermatozoa**, minute bodies with an oval or pear-shaped head, and a long slender tail. Human spermatozoa have a flattened, almost oval head, and vary in length from $\frac{1}{1000}$ to $\frac{1}{500}$ of an inch, the head being about $\frac{1}{9000}$ of an inch in diameter. Sometimes the seminal fluid contains numerous spermatozoa, at other times only a few, and frequently they are at times absent from the seminal fluid, even of young healthy men. Hence, while the discovery of spermatozoa in a stain is positive evidence of its seminal origin, their non-discovery is perfectly consistent with the same supposition. No stain, however, can positively be declared to be a stain of seminal fluid, unless **complete spermatozoa** are found therein. To detect spermatozoa in stains on cloth, &c., cut out a small portion of the stained fabric, place it in a watch glass, with enough water to thoroughly moisten it, and after half an hour or so, squeeze out the fluid and examine it under the microscope, using at least a quarter-inch objective. Spermatozoa are sometimes very difficult to separate from stains on cloth or other fabrics. Sometimes, also, stains on the garments of a female contain large quantities of epithelial scales, derived from vaginal mucus, in which spermatozoa can only be found on very careful search. Hence it must not be concluded that spermatozoa are not discoverable in a stain until several drops of the fluid obtained therefrom have been examined. An animalcule discovered in the vaginal mucus by M. Donné, and named by him Trichomonas vaginæ, can readily be distinguished from the seminal animalcule by the following characters: (*a*) Its head is at least three times as large, and is granular in the interior, not transparent as in the seminal animalcule; and (*b*) several short ciliæ are attached to its head.

Seminal stains on linen, &c., are slightly stiff to the feel,

[1] Tidy, ii., p. 211. [2] Chevers, p. 687.

greyish in colour, and become yellow when warmed; none of these characters can, however, be relied on as distinguishing seminal from other stains.

Spermatozoa resist putrefaction powerfully, and have been found in stains several months old, and even in stains three or four years old.

Evidence in Rape Cases.

1. Evidence derived from examination of the female.

On examination of the female it may be found that there are signs of—1. Defloration. 2. Other injury to the genitals. 3. Infection with disease. 4. Injuries on parts of the body other than the genitals. 5. Staining of the clothes or person with blood or seminal fluid. And the value of each of these as evidence of rape must now be considered.

1. **Signs of defloration,** *i.e.*, of loss of virginity.—These obviously are only available as evidence of rape in cases where the female was "virgo intacta" previous to the commission of the offence. Just as the presence of an intact hymen is the most reliable sign of virginity, so rupture or laceration of this membrane is the chief sign of defloration available as evidence of rape, the various other signs of virginity being, as a rule, not lost as a consequence of one intercourse. Recent lacerations of the hymen are "sharp-edged, fresh-looking, and tender";[1] and when rupture of the hymen has recently occurred, the carunculæ myrtiformes are found swollen and tender. Along with injury to the hymen, we may expect to find in recent defloration, especially if due to rape, a hot and tender condition of the genitals, accompanied by pain in walking, and pain and difficulty in passing urine and fæces, perhaps lasting some days (see *Case* C 15). There may or may not be laceration of the fourchette. Laceration or rupture of the hymen may, however, occur independently of sexual intercourse, viz., from the introduction of foreign bodies other than the penis (see *ante*, p. 317). Rape, again, even in females over the age of puberty, may be unaccompanied by injury to the hymen; and, as already pointed out, in very young children the hymen is not usually even lacerated by sexual intercourse. On the whole, therefore, the presence of signs of recent injury to the hymen, is to a certain extent evidence, but by no means conclusive evidence, in support of a charge of rape. The evidence, however, in favour of rape becomes stronger in proportion as the signs of local and other injury are greater. On the other hand, especially in young children, the absence of injury to the hymen cannot be taken as negativing the supposition that rape has been committed.

[1] Tidy's Leg. Med., ii., p. 200.

2. **Signs of other injury to the genitals.**—Rape by an adult on an immature female usually causes a considerable amount of local injury. The injury may amount simply to bruising, but frequently laceration of the parts results, and these lacerations may be extensive and severe enough to cause death. Severe injuries may be followed by inflammation and sloughing of the parts; but it must be recollected that female children are liable to a disease called noma pudendi, regarded by Mr. Kinder Wood as a form of typhus, the characteristic symptom of which is inflammation of the genitals, followed by sloughing and mortification. *Case* C 22 illustrates the appearances found in such cases. Again, as already pointed out, injuries to the genitals of immature females resembling those resulting from rape, have been caused by the introduction of foreign bodies other than the penis with the object of rendering them aptæ viris, or in order to support false charges (see *ante*, p. 317, and *Case* C 21). In females who have reached puberty, laceration of the genitals may be found if the disproportion between the size of the organs of the parties is great, or if much violence has been used. In adult females accustomed to sexual intercourse, lacerations are not likely to result from rape alone. Cases, however, are reported, where fatal laceration of the genitals has been produced in adult females after violation, by forcing foreign bodies such as sticks into the vagina. Bruises, scratches, and marks of violence, other than those caused by the introduction of the penis, may be found on the genitals, especially in adult females, but may be absent. On the whole, the presence of marks of local injury to the genitals is, to a certain extent, evidence in support of the supposition that rape has been committed. On the other hand —except when the subject is an adult female accustomed to sexual intercourse—the absence of such marks is strong, but not conclusive evidence against the same supposition.

3. **Signs of infection with disease.**—In England, India, and other countries, a popular belief exists among the ignorant that sexual connection with a virgin or young child is a certain cure for syphilis or gonorrhœa. Many cases of rape by adults on young children owe their origin to this belief; and there is no doubt but that in this way female children are frequently infected with gonorrhœa, a disease readily communicable to them. Ogston[1] mentions the case of a man who, while affected with gonorrhœa, had connection with four female children, communicating the disease to three of them. Female children, however, may become infected with gonorrhœa without intercourse. A case is recorded in which two girls, æt. respectively one and four years, became infected with the disease from using a sponge which had been used by a female suffering from it.[2] It is, however, not easy to distinguish non-syphilitic from syphilitic sores;

[1] Ogston, p. 96. [2] Med. Gazette, vol. 47, p. 744.

and impossible to distinguish a gonorrhœal discharge from a muco-purulent discharge, arising from natural causes, to which young children are subject. Further, gonorrhœa cannot be distinguished from a muco-purulent discharge, liable to arise in young females from inflammatory action, the result of local injury. Hence, in a child, the existence of sores cannot be accepted as proof of the fact that a rape has been committed, unless these are clearly shown to be syphilitic; and the existence of a muco-purulent discharge from the vagina of a young child is of but little value as evidence either of rape, or, if a rape has been committed, of the fact that the male was suffering from gonorrhœa at the time. Adult females are liable to leucorrhœa, which, although usually a mucous discharge, may become, like gonorrhœa, muco-purulent owing to ulceration of the vagina. In adult females, also, it must be noted that the existence of syphilitic sores or of gonorrhœa only proves impure connection, not rape. The period of incubation of syphilis, or of gonorrhœa, may have an important bearing in a case of alleged rape. This in gonorrhœa varies from some hours to three or four days, and in syphilis from ten to forty-four or more days.[1] Hence, if a female is seen within a few hours after it is alleged a rape has been committed, and is found to be suffering from a profuse discharge; or is seen within a few days, and is found to be suffering from syphilis; the presumption is strongly against the disease having been communicated during the intercourse represented as rape. It should further be noted that infected individuals do not necessarily by intercourse communicate either gonorrhœa or syphilis.

4. **Signs of injuries on parts of the body other than the genitals.**—Scratches, finger marks, bruises, or wounds, may be found on parts of the body other than the genitals, and the more the resistance offered the more likely are such marks to be found. Hence, therefore, such marks are likely to be more numerous if the subject is an adult female, and least numerous if a child. Even, however, in the case of an adult female, a rape may have been committed and no such marks may be found. This may happen if the offence was committed while the female was insensible, or if several persons combined in the assault, some holding the female; or even where one man only has been concerned in committing the offence, when the woman has been nearly suffocated by her clothes being thrown over her head. Marks of violence employed to prevent the female crying out, may be found on the mouth or throat. Sometimes violence employed for this purpose, results in death from suffocation. Thus Chevers mentions a case where the husband of a young girl, in order to stifle her cries during first connection, bound a cloth over her mouth, and after intercourse found her to be dead.[2] In a case which came before the Bombay Chemical Analyser's

[1] Tidy, Leg. Med., ii., p. 205. [2] Chevers, p. 695.

office, it was reported that "deceased had been violated before death, and that the cause of death was suffocation, produced by the forcible introduction of earth into the mouth and windpipe."[1] It must not be forgotten that slight injuries may be self-inflicted, with a view of supporting a false charge (for discrimination of these, see self-inflicted wounds).

5. **Staining of the clothes or person with blood or seminal fluid.**—The clothes may be found torn or stained with blood, and marks of blood may be found on the person. Of course, in such a case, the question will arise whether the blood is menstrual or not. As already pointed out, stains of menstrual blood cannot be distinguished from stains of other blood; by inquiry, however, it may be ascertained whether or no the female was menstruating at the time of the alleged commission of the offence. If the female is seen soon after the alleged rape, the discharge from the vagina, if a discharge exists, or the vaginal mucus, should be examined for the presence of spermatozoa. Spermatozoa may even be found in the vaginal mucus, ten to fourteen days after rape. Stains containing spermatozoa may be found on the clothes; but it must always be recollected that the non-discovery of spermatozoa, does not prove the absence of semen.[2]

A medical witness may, besides being called upon to examine a female for evidence of forcible intercourse, also be called upon to examine her **as to her age.** In India, the question of the age of the female will simply be: Is she under ten? In England, the question will be: (*a*) Is she under thirteen? and if not, (*b*) is she under sixteen? (See *ante*, p. 320.)

2. Evidence from the examination of the accused and of the spot where the offence is alleged to have been committed.

The examination of the accused may have to be directed towards ascertaining—1. His age, and capacity for commission of the offence. 2. Whether he is suffering from syphilis or gonorrhœa. 3. Whether his clothes or person exhibit signs of his having recently had sexual intercourse, or of having been engaged in a struggle; and the spot where the offence is alleged to have been committed may have to be examined for indications of a struggle having taken place thereon.

1. In England, the question whether the accused is over or under the age of fourteen may have to be determined. In India, the questions in regard to the **age of the accused** will be whether or no he is under seven, or, if over seven, under twelve. It may be pleaded on behalf of the accused, as a defence, that he is impotent and incapable of committing the offence. In such a

[1] Report for 1884, p. 11. [2] Tidy, ii., p. 303.

case the first question which will arise is: Does the accused appear to have attained the age of puberty? and if this be answered in the affirmative, the further question will then arise: Is there any reason to suppose that he is impotent from malformation, defect, disease, or advanced age? (See *Impotence*, p. 312.)

2. From what has been said under examination of the female **for signs of infection with disease**, it is obviously very important to ascertain whether the accused is suffering from gonorrhœa or syphilis.

3. **Stains of seminal fluid** may be found on the accused's clothes; not much weight can, however, be attached either to their presence or to their absence. If the accused is seen soon after the alleged rape, seminal stains may be found on his person, or spermatozoa may be found in his urethra. The presence of spermatozoa in this last situation, although somewhat stronger evidence of recent intercourse than seminal stains on the clothes or body, may be accounted for by spermatorrhœa, or by the occurrence of emission without penetration. **Stains of blood** on the clothes or person; or of earth, mud, &c., derived from the spot where the offence is alleged to have been committed, should be searched for. Marks of injury on the person indicative of a struggle, may or may not be present, according to the amount of resistance offered. The spot where the offence is alleged to have been committed, may show signs of a struggle having taken place, or there may be blood marks on it, or an impress of the body of the female on the ground.

UNNATURAL OFFENCES.

An unnatural offence is defined by sect. 377 of the Indian Penal Code to be "carnal intercourse against the order of nature with any man, woman or animal," and, like in rape, "penetration is sufficient to constitute the carnal intercourse necessary to the offence."

The law of England on this subject is as follows:—"Whosoever shall be convicted of the abominable crime of buggery, committed either with mankind or with any animal, shall be liable," &c., &c. (24 & 25 Vict. c. 100, s. 61). Further, from a decision in England, it would appear that to constitute the crime of "buggery with mankind" the penetration must be anal; introduction into the mouth was held not to constitute the offence. Whether introduction into the anus is necessary to constitute the "carnal intercourse with any man or woman," contemplated by sect. 377 of the Indian Penal Code, does not appear to have as yet been decided.

Writers on medical jurisprudence describe three principal forms of unnatural intercourse, viz.: 1. **Tribadism**—*i.e.*, between one human female and another. 2 **Sodomy**, *i.e.*, where the

active agent is a man and the passive agent a man or woman; this includes pæderastia, where the passive agent is a boy. 3. **Bestiality,** *i.e.*, intercourse of mankind with the lower animals.

Of these forms, tribadism can hardly be considered to come under the definition of an unnatural offence as laid down by Indian or English law.

Sodomy.

This offence appears to be largely practised in many countries, and to be very largely practised in India; indeed, Chevers mentions a case where two men, convicted of this crime on their own confession, defended themselves by putting forward the plea that "it was their occupation."[1] The offence is not uncommon in prisons, and it is a well-known prison rule that where more than one prisoner is confined in one cell, the number should never be less than three. Sometimes the offence is practised between two men, either taking alternately the part of active and passive agent. In other forms of the offence, the passive agent is a boy, and in others, again, a eunuch. In the course of a trial at Mynpoory in 1852, it came out that a great number of eunuchs regularly practising the offence exist in India; these go by the name of "*hijrahs*," dress as women, and profess to obtain their living by dancing and singing at births and marriages.[2] They recruit their ranks by castrating boys;[3] as a rule, making a clean sweep of the whole of the genital organs (see also *Injuries of the male genitals*, p. 68). It should be mentioned that in European countries false charges of sodomy are sometimes made for purposes of extortion. Where the act has been done with consent, the law regards the active and passive agent as equally guilty. In England, however, if one of the two is over and the other under fourteen, the one over fourteen alone is charged. In India, the question of age in relation to responsibility for this offence is governed by the general exceptions of the Penal Code (sects. 82 and 83, see p. 2). As in the case of rape, the question of alleged capacity of the active agent may form part of the inquiry.

Evidence of Sodomy.

1. **As to habitual practice** of the offence.—Male adults, who habitually practise sodomy, often affect effeminate manners, dress like women, &c.; and, as already pointed out, the passive agents in India are frequently eunuchs. It has been alleged that the habitual practice of the offence as an active agent gives rise to certain changes in the genital organs, *e.g.*, elongation and constriction of the penis, and twisting of the urethra.

[1] Chevers, p. 708. [2] *Ibid.*, p. 707. [3] *Ibid.*, p. 497.

On the whole, however, examination of an individual is not likely to afford any trustworthy indication as to whether or no he habitually practises sodomy as an active agent. Where the individual has been habitually practising the offence as a passive agent, the skin around the anus may be found to have assumed a smooth appearance, instead of showing the usual series of folds extending "concentrically towards the anal aperture."[1] This, Casper considers to be "the most certain of all the uncertain signs of passive pæderastia."[2] There also may be a "funnel-like" or "trumpet-shaped" depression of the nates leading to the anus. The presence of a chancre about the anus, or of a gonorrhœal discharge from the rectum, is, of course, strong evidence that the individual has been the passive agent in the offence, and may be corroborative evidence of his having acted in that capacity.

2. As to recent commission of the offence.—If it is alleged that the offence has been recently committed without consent, both parties should be examined for marks of violence indicative of a struggle, as in a case of alleged rape; and whether the act has been done with consent or not, the alleged active agent should be examined in the same way as the accused in a rape case. Examination of the passive agent may show stains of blood, or seminal fluid, on his clothes, or person in the neighbourhood of the part; or, if the individual is a young boy or a person unaccustomed to the offence, there may be found about the anus bruising or excoriations of the mucous membrane, or, perhaps, slight laceration of the sphincter.[3] Obviously also, the question of the age of the parties must be considered, seeing that this bears on the question of their criminal responsibility.

Bestiality.

The form of this offence in which a human male is the active agent, is tolerably frequently met with in India. Chevers

[1] Casper, iii., p. 333. [2] *Ibid.*, p. 334.

[3] Dr. J. W. Johnstone (Chevers, p. 708), with reference to the physical evidences of sodomy, states, "Penetration seldom reaches beyond an inch, and the force expends itself on the semi-lunar folds which in the empty gut droop on either side. One arc of the lower anal mucous fold occupies the superior and upper left, a second the inferior lower and right angle of the rectum, and hangs down in a central crescent; the foreign agent impinges against this loose centre, stretches its point of attachment at one or other of the angles, and in every case of clear penetrative contact (provided the gut has not been distended by polypi, as is not infrequent in children, or by systematic sodomy, when the mucous membrane loses its rugæ and the sphincters their contractile power) rupture will be found cutting horizontally outwards at the left superior or right inferior angle.... The shape of the wound is characteristic, and it cannot be produced by any hard substance. A true sodomy wound is triangular; the base external, with the sides of the triangle retreating into the fundament." He (Dr. Johnstone) "has never observed any other portion of the mucous membrane ruptured by a specific act of sodomy."

mentions an instance of its commission with a goat, and others of its commission with mares. I have had to examine matters for the presence of spermatozoa, in cases where the offence, it was alleged, was committed with respectively an ass, a cow, and a hen.

In these cases, matters removed from the vagina of the animal with which the offence has been committed, or adhering to the surrounding hairs, may have to be examined for the presence of spermatozoa. Detached hairs also may be found adherent to the person or clothes of the accused, and may have to be examined as to the identity or otherwise of their appearance, with the hairs of the animal employed as the passive agent.

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CHAPTER XXVIII.

PREGNANCY.

Cases involving the determination of the existence or non-existence of pregnancy.

There are five principal cases in which the existence, or otherwise, of pregnancy may have to be determined, viz.:—

1. **When pregnancy is pleaded in bar of execution** (see *ante*, p. 3). In such a case in India, owing to the wording of sect. 382 of the Code of Criminal Procedure, the question to be decided is simply, Is the woman pregnant or not? In England, however, owing to the terms of the charge to the jury of matrons, a medical man called in to their assistance may have to examine into the further question, Is the woman "with child (pregnant) of a quick child"?

2. **Where a widow is suspected of feigning pregnancy in order to ultimately produce a supposititious heir** to an estate of which her husband died possessed.—In such a case, according to the law of England, the heir-presumptive to the estate, *i.e.*, the person who would succeed thereto, supposing the woman not to be pregnant, may apply to the Court to order an inquiry to be made into the alleged pregnancy. The Court, if it grants the application, does so by issuing what is technically called a writ "de ventre inspiciendo."

3. **Where the question is whether or no a woman is pregnant as the result of adulterous intercourse.**—The woman alleged to be pregnant may be a married woman living apart from her husband, and the allegation may be put forward in support of a suit for divorce. Or she may be an unmarried female or a widow, who has been defamed.

4. **Where the existence of pregnancy supplies a motive** for murder or suicide (see *Cases* C 23 to 26).

5. **In cases of alleged causing, or attempting to cause, miscarriage.**—In such cases, as the fact of the existence of pregnancy may support the allegation that an attempt has been made to cause miscarriage, the question whether or no a certain female is pregnant may arise. It should, however, be noted that an attempt to cause miscarriage is an offence, irrespective of whether the woman be or be not pregnant. Further, according

to the law of India (but not according to that of England), to cause, or attempt to cause, a woman "quick with child" to miscarry, is a graver offence than if she be not quick with child.[1] Hence, in India, in these cases the question may arise, whether or no a certain female was "quick with child" at a particular time (see *Causing miscarriage*).

We have therefore to consider—I. What are the evidences of the existence of pregnancy? and II. What is the meaning of the phrases "with child of a quick child," and "quick with child"? and how may the existence or otherwise of the condition thus defined be determined?

I. What are the evidences of the existence of pregnancy?

It is convenient to divide these as follows:—1. The statement of the woman as to her condition. 2. Signs of pregnancy not directly referable to the fœtus. 3. Signs of pregnancy directly referable to the fœtus and its appendages.

1. **The statement of the woman as to her condition.** This obviously cannot be relied on for forensic purposes. Apart, however, from any wilful endeavour to deceive, a woman may be mistaken as to her condition. She may mistake, for example, symptoms of organic disease for symptoms of pregnancy. Cases also are recorded where, no organic disease being present, symptoms closely simulating those of pregnancy (spurious pregnancy), and, in exceptional cases, of labour also, have appeared. Again, a pregnant woman attributing her symptoms to disease, may be unaware of her condition, and remain so, even up to the time of her delivery. Further, as impregnation is independent of volition on the part of the female, conception may occur as the result of intercourse effected with her while in an insensible condition, and in such a case a woman may be unconscious of the fact that she is pregnant, and, it is possible, remain so up to the time of her delivery.

2. **Signs of pregnancy not directly referable to the fœtus.**

a'. **Cessation of menstruation.**—This sign is open to several fallacies. Menstruation may cease owing to causes other than pregnancy. A discharge of blood simulating menstruation may occur during pregnancy. Again, a woman may feign or deny menstruation in order to conceal her condition.

b'. **Constitutional disturbance.**—Pregnancy is often accompanied by signs of constitutional disturbance, especially of the function of digestion, morning sickness being a common symptom. Similar symptoms may, however, arise from causes other than pregnancy.

c'. **Changes in the mammæ.**—The breasts enlarge, become

[1] Indian Penal Code, sect. 312.

firmer, and secrete milk. A dark circle (areola), varying in width from half an inch to three inches, studded with glandular follicles, develops around the nipple. These appearances may, however, arise from causes other than pregnancy, or may continue after delivery. Hence they may be present in a non-pregnant female. Again they may be absent in pregnancy.

d'. **Kyestin or gravidine in the urine.**—This appears in urine, after it has stood for some time, as a fatty pellicle on its surface. Its formation is usually, but not invariably, observed in the urine of pregnant females after the first month. It has, however, been seen in the urine of men, and in the urine of non-pregnant females. But little weight can be attached to its presence or absence.

e'. **Changes in the uterus.**—The cervix becomes full, round, soft, and elastic, and the os loses its transverse shape, and becomes circular, and its edges become soft and indistinct. Up to the end of the third month, the uterus not having risen out of the pelvis, the cervix is low down in the vagina and easily reached, and no enlargement of the abdomen is perceptible. After this the uterus begins to rise, and the cervix to shorten, recede, and become indistinct. About the end of the fourth month the enlarged uterus begins to be perceptible above the pubes, and rises to—between the pubes and umbilicus during the fifth month; the umbilicus during the sixth month; half-way between the umbilicus and the lower end of the sternum during the seventh month; and to the ensiform cartilage during the eighth month. As similar changes may take place owing to enlargement of the uterus from causes other than pregnancy, more reliance is to be placed on their absence as a negative sign, than on their presence as a positive sign of pregnancy.

3. Signs of pregnancy directly referable to the fœtus and its appendages.

a.' **Detection of the active movements of the fœtus.**—If the hand, especially after immersion in cold water, be placed on the abdomen over the uterus, the movements of a living fœtus within its cavity may be felt, in some cases as early as during the fourth month, in others not until the fifth month, or later. This sign is, of course, only available if the fœtus be living. It is open to several fallacies: thus the movements may not be detected until after several examinations have been made; or, in exceptional cases, may not be detected at all during pregnancy. Again, movement of flatus in the intestines, or contractions of the uterus, or of the abdominal muscles, may be mistaken for the movements of a fœtus.

b'. **Detection of passive movement of the fœtus** (Ballotement).—This sign is not available until the end of the fourth month. It consists in the detection of a solid body floating (in

the liquor amnii) in the uterus. To obtain it, the woman—her bladder and rectum having been previously emptied—should be placed in the upright position; or recumbent, with the shoulders much raised. One or two fingers of one hand are then to be introduced into the vagina, and applied to the point of the cervix. The other hand is steadily pressed on the abdomen over the uterus. A jerk upwards is then given with the fingers in the vagina, when a hard body will be felt to recede from, and in three or four seconds fall back on, the fingers. After the end of the sixth month this sign is rarely available, the fœtus, from its bulk, not floating freely enough in the liquor amnii. Care must be taken to keep the fingers in contact with the cervix, otherwise a movement of the uterus itself may be mistaken for the movement of the fœtus within it.

c'. **Detection of the sounds of the fœtal heart.**—This sign Guy and Ogston describe as resembling the ticking of a watch heard through a pillow. The pulsations vary from one hundred and twenty to one hundred and sixty a minute, and are **not synchronous with the mother's pulse.** A double sound is heard at each pulsation. The sounds are generally, but not always, heard about midway between the umbilicus and one or other of the anterior superior spines of the ilium; they seldom can be heard earlier than the end of the fifth month. If detected, the existence of pregnancy is certain, but pregnancy may exist and the sounds not be detected (*a*) owing to the examination being made at too early a stage; (*b*) owing to the death of the fœtus; or (*c*) owing to want of skill on the part of the auscultator. Ogston records a case where owing to ascites the sounds could not be detected by a skilled auscultator.

d'. **Detection of the uterine murmur.**—This, generally attributed to the flow of blood through the placenta, is described by Guy and Ogston as a low cooing sound, such as is made by blowing gently over the lip of a wide-mouthed phial. It is **synchronous with the mother's pulse,** and is first distinctly heard about the end of the fourth month. This sound is liable to intermission, and, according to some writers, may not be heard at all in pregnancy, if the placenta is attached to the posterior wall of the uterus. Other sounds also may be mistaken for it, *e.g.*, the muscular susurrus of the uterus, the bruit of a vascular sarcoma of the uterus, or a bruit resulting from pressure on the abdominal aorta.

In cases where the examination is conducted after death, the discovery of an ovum or fœtus in the uterus is, of course, an unequivocal sign of the existence of pregnancy. For the characters of the ovum or fœtus at various stages of gestation, see *Appendix, Tables XVIII. to XX.* The presence also of a corpus luteum in the ovary, may afford corroborative evidence (see p. 381).

II. What is the meaning of the phrases "with child of a quick child," and "quick with child"? and how may the existence or otherwise of the condition thus defined be determined?

It may first be remarked that the term "**quickening**" is applied to certain peculiar sensations experienced by the mother at a certain stage of pregnancy. These sensations are often accompanied by constitutional disturbance, and are popularly ascribed to the first perception by the mother of the movements of the fœtus. They are most probably due to this cause, perception of the movements probably first occurring when the uterus comes into contact with the abdominal wall. These sensations may be felt as early as the twelfth week, are generally first felt between the fourteenth and twenty-fourth week, but in some cases are not felt at all during pregnancy. A woman who has felt these sensations is said to have quickened. These two terms, "quickening" and "quickened," are derived from the word "quick," used in its old signification, viz., living. Their use with reference to these sensations, arises from the old popular belief that their occurrence denoted the first accession of life to the fœtus. As, however, a fœtus is actually alive from the moment of conception, two interpretations may be assigned to the word "quick" when applied to a fœtus in utero: (1) The more extended interpretation, viz., that the fœtus is alive; or (2) The more restricted interpretation, viz., that the mother has experienced the sensation known as quickening.

As regards the first of the two phrases in question, viz., "with child of a quick child," there appears to be no doubt but that this has always been used in law as if the more restricted meaning attached to the word "quick." Some doubt, however, has been thrown on the interpretation accepted by English legal authorities of the second phrase, viz., "quick with child," owing to the remarks made by Baron Gurney in the case of *R.* v. *Wycherley* (8 C. & P. 262). In this case pregnancy having been pleaded in bar of execution, the jury of matrons were directed to try whether the prisoner was "quick with child or not." Subsequently Baron Gurney addressed a medical witness called to the assistance of the jury of matrons, as follows: "Quick with child is having conceived; with quick child is when the child has quickened. Do you understand the distinction?" Baron Gurney, therefore, in this case directed the medical witness to take the expression "quick with child" as if the more extended meaning attached to the word "quick." This, however, is contrary to the law as stated by Blackstone, who says: "If they (the jury of matrons) bring in their verdict quick with child—for barely with child, unless it be alive in the womb, is not

sufficient—execution shall be staid." . . . "But if she (the prisoner) once hath had the benefit of this reprieve, and been delivered, and afterwards become pregnant again, she shall not be entitled to the benefit of a further respite for that cause. For she may now be executed before the child is quick in the womb." In the Indian Penal Code also, the expression "quick with child" is clearly used as if the more restricted meaning attached to the word "quick." Sect. 312, for example, makes causing miscarriage, if the woman be "quick with child," a graver offence than simply causing miscarriage, thus implying that the condition "quick with child" is one which arises at a period subsequent to conception.

When, therefore, the question arises, is a certain woman "with child of a quick child" (or "quick with child"), what has to be determined is whether or no the woman has quickened. Quickening, however, is a sensation only felt by the mother. Still, if a medical man has, on examination, felt the actual movements of the fœtus, he is justified in assuming that the mother has also felt them, and that therefore she has quickened. Should he be unable to detect the active movements of the fœtus, he can only, in answer to the question, Has this woman quickened? state his opinion as to whether or no (1) The woman is pregnant; (2) The child is alive; and (3) The pregnancy has advanced to, or beyond, the stage at which the sensation of quickening is usually experienced; leaving it for the Court to decide whether his answers do or do not amount to an affirmative answer to the question, Has this woman quickened? In giving an opinion on the last of the three above-mentioned points, a medical witness should bear in mind that quickening does not occur at any fixed period. As before stated, it may occur at any time between the twelfth and twenty-fourth week. Further, it may be noted, that of the two cases in which the question of quickening arises, viz., the English case of pregnancy pleaded in bar of execution, and the Indian case of causing miscarriage; in the first the prisoner is benefited by being found "quick with child," while in the second a similar answer has the reverse effect.

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CHAPTER XXIX.

DISPUTED RIGHT TO INHERIT.

In certain cases where the right to inherit property or a title is in dispute, medico-legal questions may arise. Thus, when the succession is fixed on the male to the exclusion of the female line, the question may arise, Of what sex is a certain individual? (See *Sex.*) Again, as by law children born "without the shape of mankind" cannot inherit, the question may arise, Has this child "the shape of mankind"? On this question, however, no general rules can be laid down. More important cases are those where the right to inherit is disputed, on one or other of the following grounds:—

1. That the claimant is not a legitimate child; and with the medico-legal questions which arise in such a case, we may consider those which arise in affiliation cases.

2. That, as in tenancy by courtesy cases, a certain child was not born alive.

3. That the claimant is a supposititious child.

The following remarks will show what medico-legal questions may arise in the above cases.

1. **Legitimacy.**—Children are either legitimate or illegitimate; the latter are also called "bastards." Only legitimate children are regarded by law as the children of their father. These, therefore, possess certain rights which illegitimate children do not possess. According to the law of most countries, only such children are held to be legitimate, as are either born or begotten during the existence of a valid marriage (lawful wedlock) between their parents. By the law of Scotland, however, children born before marriage, become legitimate on the subsequent marriage of their parents. Further, according to the **law of England**, any child born or begotten during lawful wedlock, is presumed to be legitimate until the contrary is shown; (*a*) by proof of the impotence of the alleged father of the child; or (*b*) by proof that the parties to the marriage had no access[1] to each other at any time when the child could have been begotten. The presumption, as to legitimacy, of the **law of India** is embodied in sect. 112 of the Indian Evidence Act, and is as follows: "The fact that any person was born during the continuance of a valid marriage between his mother and any man, or within two

[1] See note, Appendix XVII., on "Access."

hundred and eighty days after its dissolution, the mother remaining unmarried, shall be conclusive proof that he is the legitimate son of that man, unless it can be shown that the parties to the marriage had no access[1] to each other at any time when he could have been begotten."

Hence, the **legitimacy** of a child **may be disputed** on either of two grounds, viz. (1) on the ground that the alleged father of the child is **impotent**; or (2) on the ground that the parties to the marriage had **no access** to each other at any time when the child could have been begotten. The following examples show the medico-legal questions which may arise when legitimacy is disputed on the second of these two grounds.

(1) A husband on a certain date ceases to have access to his wife; after a certain interval the wife is delivered of a child. In such a case the legitimacy of the child may be disputed, on the ground that the interval between the last access of the husband and the birth of the child, was greater than the utmost period to which gestation can be prolonged.

(2) The parties to a marriage are proved, after a long period of separation, to have resumed access to each other on a certain date. After the lapse of a certain interval the wife is delivered of a child. In such a case the legitimacy of the child may be disputed, on the ground that the period intervening between the date of resumption of access and the date of the child's birth, was so short, that the child must have been begotten before access was resumed If in such a case the appearance of the child at birth indicates it to be a mature child, the question arises, What is the shortest natural period of gestation? or if the child is an immature child, what, judging from its appearance, was its uterine age at the time of its birth? Again, in such a case it may be alleged that the mere fact that the child was born alive and capable of being reared, proves that its uterine age at birth was greater than the interval which elapsed between resumption of access and birth, thus raising the question, What is the earliest period of gestation at which a "viable" child can be born, *i.e.*, one capable of living and being reared? Moreover, as a portion of the evidence bearing on the question of early viability, is derived from cases where a viable child has been born a short time after a previous delivery, and as such cases may be accounted for by "superfœtation" (*i.e.*, conception of a second ovum during gestation of a first), the further question arises, Is superfœtation possible?

It may be here remarked that as his wife's adultery is a ground on which a husband may claim a divorce, questions similar to those arising in cases of contested legitimacy, may arise in suits for divorce. The question as to the degree of maturity of a child, may also arise in cases where a child is born

[1] See note, Appendix XVII., on "Access."

soon after marriage, and where it is alleged that the parents must in consequence have had sexual intercourse before marriage, and are therefore of immoral character (see *Case* C 27).

2. **Affiliation cases.**—Although illegitimate children are regarded by law as the sons of nobody, their father is bound to contribute towards their support until they have attained a certain age. Hence, a woman having been delivered of an illegitimate child, may appear before a Court and claim that a certain individual, who she alleges is the father of her child, may be compelled to so contribute. Such cases are called "affiliation cases," and in them questions may arise, similar to those arising in cases of contested legitimacy. In affiliation cases, also, the further question may arise, Can any opinion as to the paternity of the child be formed from its resemblance or non-resemblance to its alleged father?

3. **Tenancy by the courtesy of England.**—By the common law of England, if a man survived his wife, and he had issue by her born alive, that might by possibility inherit the estate as her heir, the husband so surviving became entitled to an estate for the residue of his life in such lands and tenements of his wife as she was solely seised of in fee simple, or fee tail in possession. The husband, while in enjoyment of this estate, is called a tenant by the courtesy of England, or, more shortly, tenant by courtesy.[1] To establish this tenancy by courtesy, the child must be born during the existence of the marriage, and hence, although the right accrues to the husband if the child is extracted by Cæsarean section during the mother's life, it does not accrue if the child is so extracted after her death, for in that case the marriage has ceased to exist before the birth of the child. Further, the child must have been completely born, and must, after complete birth, have manifested some sign of life; the slightest sign of life, however, a mere tremulous motion of the lips for example (see *Case* C 28), has been held by the English Courts sufficient to establish the fact of live birth in these cases.[2] Much stronger evidence of live birth is, however, required in cases of infanticide (see *Infanticide*). As in a case of disputed right to tenancy by courtesy, it may be alleged in opposition to the claim, that the child when born was so immature, that it could not possibly have manifested any sign of life after birth; the question may arise in these cases, What is the earliest period of gestation at which a child can be born, capable after birth of manifesting signs of life?

It is possible that a claim to be tenant by the courtesy might arise in India, as there are estates held in India subject to the

[1] Williams on Real Property, p. 274.

[2] The Scotch Courts require, in order to establish the fact of live birth in civil cases, proof of commencement of respiration (Ogston, p. 182). For crying as a proof of birth, see Infanticide, p. 354.

English Law of Inheritance (see remarks of Mr. Justice Pontifex in *Case* C 29). No such claim, however, could be made by any one whose marriage had taken place since the 31st of December, 1865, as sect. 4 of the Indian Succession Act, 1865, enacts that no person shall by marriage acquire any interest in the property of the person whom he or she marries. That section by sect. 331, is not applicable to marriages contracted before the 1st of January, 1866.

4. **Supposititious children.**—By a supposititious child is meant a child produced by a woman who avers it to be hers when it is not. In these cases the motive is generally to further an attempt, either to extort money, or to divert succession to property. A supposititious child may be one (1) produced by a woman who has never been delivered of a viable child; or (2) produced by a woman in substitution for a child of her own. In case (1), besides questions similar to those occurring in legitimacy cases, the following additional questions may arise: (*a*) Is this woman sterile? and (*b*) does this woman show signs of having been recently or previously delivered of a viable child? In case (2) it is very seldom that medical evidence can afford any assistance. In both cases, as in affiliation cases, the question of how far the paternity of a child can be inferred from its resemblance or non-resemblance to its alleged parents may also arise.

Hence the following are the principal medico-legal questions which may arise in cases of disputed right to inherit:—

I. Is a certain individual impotent or sterile? This question has already been considered (see *Impotence and Sterility*, p. 310).

II. What is the natural period of human gestation?

III. How far may this period be prolonged?

IV. Is superfœtation possible?

V. What is the earliest viable age?

VI. What are the characters of children born at various periods of gestation?

VII. How far may the paternity of a child be inferred from its resemblance or non-resemblance to its alleged parents?

VIII. Has this woman ever been delivered of a viable child?

Question II.—What is the natural period of human gestation?

The two most reliable methods of estimating the duration of gestation are: (1) By observation of the period intervening between cessation of menstruation and delivery; and (2) by observation of the period intervening between a single coitus and delivery. Of these two methods the first cannot be relied on to give precise results, because—

(*a*) Menstruation may cease from causes other than pregnancy, or may continue after pregnancy has commenced; and

(*b*) Impregnation may occur at any period during the menstrual interval.

The second method, although more precise than the first, also cannot be relied on to give accurate results, because impregnation is not necessarily coincident with coitus, but may occur as long thereafter as the spermatozoa retain their vitality, which they may do for several days after emission.

Even, however, when the second and more accurate of these two methods is employed, results varying considerably are obtained, from which it appears that the duration of natural gestation is **not a fixed period**, but one subject to variation within certain limits. Guy, for example, states that of fourteen authentic cases in the human subject, in which the duration was ascertained by reckoning from a single coitus, the minimum duration was 270, the maximum 293, and the average 284 days.[1] Again, Wharton and Stillé give a table of all the authentic cases of this kind in the human subject they have been able to collect.[2] Their table includes fifty-six cases, and shows a range of duration of from 260 to 296 days, with an average of 276 days.

The view that the **duration of pregnancy is not a fixed period** is **supported** by the results of **observations on the lower animals.** Thus from three series of observations on cows, the minimum period in these appears to be 260 days, and the average period 280 to 285 days: but in one series[3] (160 animals) a period of 308 days was observed; in the second series[4] (760 animals) a period of 313 days was noted; and in the third[5] (1105 animals) in four delivery took place in the forty-eighth week, equal to a duration of over 329 days; and in one in the fifty-first week, equal to a duration of over 350 days. Again, a series of 102 observations on mares[6] gave a range of 311 to 394 days, with an average of about 340 days; and another on 177 sheep,[7] a duration of 145 to 171 days, with an average of 150 days.

Question III.—How far may the period of human gestation be prolonged?

The chief considerations bearing on this question are as follows:

1. Of the fifty-six authentic cases collected by Wharton and Stillé, in which the duration of gestation was fixed from a single coitus, in nineteen the duration was over 280 days, and in two of these it was 291, and in three others 296 days.

[1] Forensic Medicine, p. 127.
[2] Med. Jurisp. (1884), iii., p. 41.
[3] Tessier's, Guy, p. 128.
[4] Earl Spencer's, *ibid.*
[5] Krahmer's, Wharton and Stillé, iii., p. 44.
[6] Tessier's, Guy, p. 128.
[7] Krahmer's, Wharton and Stillé, iii., p. 43.

2. In exceptional cases, where the commencement of pregnancy has been fixed by the death or absence of the husband or male, a longer period than 296 days has been recorded. Thus Guy, on the authority of Hewitt, quotes a case in which the duration of pregnancy, as fixed by the sudden death of the husband, was 308 days; and in two less satisfactory American affiliation cases, in which the commencement of pregnancy was fixed by date of last intercourse, the alleged duration was respectively 313 and 317 days. In both these cases the Court decided in favour of the plaintiff, thus admitting the possibility of prolongation of pregnancy to the periods stated.

3. In a very large number of cases recorded by various authorities, in which the duration of pregnancy was estimated from the last day of menstruation, the longest period recorded was 325 or 326 days. As, however, conception may occur at almost any period during a menstrual interval, these cases cannot be relied on as showing anything more than that pregnancy may be prolonged for 325 or 326, less (say) 23 days. This would give 303 days, or a shorter period than in Hewitt's case. In four less certain cases of the same kind, the estimated period of gestation was 309 and 313 days (Simpson), and 314 and 324 days (Murphy).

4. In the lower animals, it has been observed that the duration of pregnancy, as estimated from a single coitus, may be greatly protracted beyond the usual period.

On the whole, therefore, as regards the question, What is the longest period which in natural human gestation may intervene between coitus and delivery?—the form which the question under consideration assumes for forensic purposes,—it may be stated that—

1. It may be regarded as proved that this may be 296 days.

2. Most authorities agree in considering that the interval may be as long as 44 weeks, or 308 days. Indeed, in the Gardner Peerage case, several eminent obstetricians gave it as their opinion that the interval might extend to, at any rate, 311 days.[1]

3. Some authorities consider that the interval may extend to the forty-sixth week, 315 to 322 days.[2]

Question IV.—Is superfœtation possible?

It may first be stated (1) that two closely following acts of intercourse in the same female may each prove fruitful (see *Case* C 30); and (2) that it cannot be doubted but that conception may occur during pregnancy in cases where the uterus is double or bipartite, a rare condition in the human female, but still one

[1] In this case the question at issue was as to the legitimacy of an individual born 311 days after the last access of the husband.

[2] See Ogston's Lectures, p. 189.

of which several instances are recorded. Excluding these two classes of cases, and limiting the question to whether, the organs of the female being of normal formation, it is possible for conception of a second embryo to occur during gestation, we find that authorities are divided in opinion on the subject. The arguments for and against the possibility of conception occurring under the conditions stated are founded on (1) physiological considerations, and (2) recorded cases.

1. **Physiological considerations.**—Those who deny the possibility of the occurrence, allege that the plugging of the os uteri and Fallopian tubes, and the formation of the decidua, events which occur at a very early stage of pregnancy, offer an impassable barrier to the passage of the seminal fluid. On the other hand, those who affirm the possibility of superfœtation, deny that these conditions invariably offer an impassable barrier to the seminal fluid (especially previous to the end of the third month[1]), and point out that, as in exceptional cases menstrual blood finds its way out of the uterus during pregnancy, it is by inference also possible that seminal fluid may find its way in.

2. **Recorded cases.**—The cases brought forward in support of the view that superfœtation is possible, may be divided into two classes, viz.:—

(*a*) Cases in which a woman is delivered at or about the same time of a more or less mature child, and a less developed dead fœtus: *e.g.*, as in a reported case, of a mature child; and a dead fœtus of apparently five months. Many cases are, however, reported showing that a dead fœtus may be retained in the uterus until the full term of pregnancy has expired, or even for a considerable period beyond. Hence cases of this class can be explained on the supposition, that conception of the two children occurred at the same time, but that one died and was retained in utero until the delivery of the other. Obviously, therefore, such cases do not support the view that superfœtation is possible.

(*b*) Cases in which a woman is delivered of two more or less mature children; a considerable interval, but still an interval shorter than the usual period of gestation, separating the two births. Cases of this kind where the interval between the births is comparatively short, *e.g.* in one reported case a month, are easily explained on the supposition, that conception of the two children occurred at the same time, but that the delivery of one was delayed. Other cases of this description again, in which the interval between the two births is comparatively long, can be explained by supposing that conception of the second child occurred, after delivery of that first born. It should, however, be noted, as bearing on this possibility, that it is highly improbable

[1] It is not until the end of the third month that the decidua reflexa, or portion of the decidua surrounding the ovum, comes into contact with the decidua vera, or portion of the decidua lining the uterus.

that conception can occur until a week after delivery; probably a fortnight must intervene.[1] A few cases, however, are on record in which the interval separating the births of two viable children has been four to five months; *e.g.*, *Case* C 32, and a case referred to by Taylor, in which the interval was 127 days (see also *Case* C 31, in which the interval was 167 days, but in which no sexual intercourse took place until twenty days after the first delivery). **Cases such as these involve the acceptance of one of three propositions**, viz., either

(1) That superfœtation is possible, even, as in case C 32, when the uterus is not double; or

(2) Supposing conception of the second child to have taken place after the birth of the first; that a viable child may be born at a very early uterine age; *e.g.*, in Taylor's case at 127, or more probably 120 days; or

(3) As suggested by Wharton and Stillé; that in cases of twin pregnancy the pressure of one child on the other, instead of, as is sometimes the case, causing the death of one of the two, may in exceptional cases simply retard its development; the result being that one child is born mature at the full period, and after its birth, development of the second child continues, until it also reaches maturity, when its birth takes place.

Question V.—What is the earliest viable age?

I.e., what is the earliest period of gestation at which a child may be born, capable of living and being reared?—Here it may first be remarked (1) that there is no doubt but that a child born at or after the 210th day of uterine life may be reared; and (2) that the evidence afforded by recorded cases so strongly supports the view that children born as early as the **180th day may be reared**, that the possibility of this cannot be denied. As regards the question of **viability before the 180th day**, it should be noted that the validity of the evidence afforded by cases cited to prove early viability, mainly depends on the accuracy with which the date of conception is determined; for although the characters of a child at birth afford indications of its age, they cannot be relied on, except as corroborative evidence. **In some** of the **cases** cited as evidence of early viability, the **date of conception is fixed from a previous delivery**; *e.g.*, the case mentioned by Taylor (see *Superfœtation*), in which a viable child was born 127 days after a previous delivery, and another similar case referred to by the same author, in which the interval between the births was 174 days.[2] If we assume that in these cases conception of the second child did not take place until after the birth of the first, we must admit viability to be possible at respectively the 120th

[1] Bonnar, Edin. Med. Journal, vol. x, p. 582. [2] Taylor, ii., p. 229.

and 167th day of intra-uterine age. Obviously, however, the acceptance of cases such as these, as valid evidence of early viability, rests on the assumption that it is impossible for either superfœtation, or retardation of development as suggested by Wharton and Stillé, to occur.

Of the cases in which the date of conception is fixed independently of a previous delivery, there is one, Dr. Outrepont's case (see *Case* C 33), in which a viable child was born twenty-five weeks (175 days) after the last menstruation of the mother. Guy, in reference to this case, says, "It is very valuable, for it is the only quite unequivocal instance on record of the rearing of a six months' child."[1] The Jardine case (*Case* C 27) is also a fairly authentic case of the rearing of a 174-day child. **There are also a few less reliable cases** of the rearing of children born at a period **earlier than the 174th day.**[2] Among these, the earliest viable age recorded is 133 days (Dr. Rodman's case).[3] The evidence afforded by these cases, in favour of viability at a period earlier than the 174th day, is further supported by certain recorded cases, in which children born at an earlier age than this, lived for some days after birth.[4]

As regards the further question, What is the earliest age at which a child may be born, capable after its birth of showing signs of life? it may be stated, that there is more than one reliable case on record, showing that a child born between the fourth and fifth month of uterine life may after birth manifest signs of life. Among these may be mentioned Dr. Barrow's case of a child born at 144 days, which after birth breathed convulsively at intervals, for forty minutes.[5]

Question VI.—What are the characters of children born at various periods of gestation?

For characters previous to the end of the fourth month, see *Appendix XVIII.* After the fourth month the uterine age of the fœtus is indicated by the following characters. (A) **During life:** (1) Its length and weight; (2) certain changes about the eyes; (3) the appearance of the skin, nails, and scalp hair; and (4) the position of the middle point of the body. (B) **After death** the following additional characters become available:—(1) The progress of ossification; (2) the condition of the intestines; (3) the

[1] Guy's For. Med., p. 136.

[2] *E.g.*, Dr. Barker's case, 158 days (Med. Times, 1850, vol. ii., pp. 249, 392), and Capuron's doubtful case of Fortunio Liceti, 135 days (Guy's For. Med., p. 134).

[3] Guy's For. Med., p. 134.

[4] *E.g.*, Fleischmann's case of a child of 168 days living for eight days (Guy, p. 134), and Dr. Routh's case of a child born between the fifth and sixth month living for eighteen days after its birth (Obstet. Trans., 1871, p. 182).

[5] Wharton and Stillé, vol. iii., p. 51.

condition of the gall-bladder; (4) the position of the testicles; and (5) miscellaneous characters. According to Guy, Tidy, and others, these characters are as follows:—

1. **The length and weight.**—The following table gives the average length in inches, and average weight in pounds and ounces, at the end of each month:—

Month	Length		Weight	
	From	To	From	To
			lbs oz	lbs oz.
4	4½	8½	0 3	0 7
5	6½	10½	0 5	1 1
6	8	13½	1 0	2 2
7	11	16	2 0	4 5
8	14	18	3 4	5 7
9	16	20	4 5	7 0

Exceptional cases are recorded of children at birth being unusually large and heavy. The greatest length and weight recorded appears to be 32 inches, and 18 lbs. 2 oz.; next to this comes a case where the length was 24 inches, and the weight 17 lbs. 12 oz.[1]

2. **Changes about the eyes.**—The eyelids are adherent, and the membrana pupillaris vascular and distinctly visible, up to the end of the sixth month. At the end of the sixth month, the eyebrows and eyelashes are beginning to form. At the end of the seventh month, the eyelids are non-adherent, and the membrana pupillaris is beginning to lose its vascularity, and by the end of the eighth month, it is so thin and transparent as to be only with difficulty discernible.

3. **Appearance of the skin, nails, and scalp hair.**—Up to the end of the fifth month, **the skin** is destitute of fibrous structure and sebaceous covering. At the end of the sixth month, it begins to show a fibrous structure, and papillæ begin to appear; at this period it is covered with down, and sebaceous matter begins to be visible on its surface. At the end of the seventh month, it is dusky red, thick, and fibrous, and covered with sebaceous matter. By the end of the eighth month, it is covered with fine short hairs, and the sebaceous envelope is well marked. At the end of the ninth month, the down has disappeared from the surface of the body except the shoulders. **The nails** begin to appear at the end of the fourth month, are very distinct at the end of the fifth month, and gradually increase in length, reaching the ends of the fingers at the end of the eighth month. **Hair on the scalp** begins to appear at the end of the fifth month, is about quarter of an inch long at the end of the seventh

[1] Taylor, ii., p. 314.

month, and at the end of the ninth month has attained a length of about an inch.

4. **The position of the middle part of the body.**—This up to the end of the fifth month lies on the body of the sternum; gradually descending, it reaches the lower end of the sternum at the end of the sixth month, is nearer the umbilicus than the sternum at the end of the eighth month, and at the end of the ninth month is generally about three-quarters of an inch above the umbilicus.

Further signs available after death.

1. **The progress of ossification.**—At the end of the fourth month the ossicles of the ear are found ossified, and points of ossification have just appeared in the upper part of the sacrum (for points of ossification appearing earlier than the end of the third month, see *Appendix XVIII.*). By the end of the fifth month, points of ossification have appeared, in the pubis, os calcis, axis, and odontoid process; at the end of the sixth month, in the four divisions of the sternum; at the end of the seventh month, in the astragalus; at the end of the eighth month, in the last sacral vertebra; and at the end of the ninth month, in the **lower epiphysis of the femur.** This last point of ossification is not present at the end of the eighth month, and great weight is attached to it by Casper and others, **as a sign of maturity.** According to Casper, its diameter in mature children is three-quarters of a line to four lines, and Tidy adds that if it is more than three lines in width, the child has probably survived its birth. "This nucleus appears to the naked eye as a more or less circular blood spot in the midst of milk-white cartilage." [1]

2. **The condition of the intestines.**—At the end of the fourth month, the duodenum contains meconium, the cæcum is placed near the right kidney, and the cæcal valve is visible. At the end of the fifth month meconium of a yellowish green tint is present at the commencement of the large intestines. At the end of the sixth month in the large intestine sacculi begin to appear, and meconium is present in the upper part. At the end of the seventh month the cæcum lies in the right iliac fossa, the valvulæ conniventes begin to appear, and meconium is present nearly throughout the whole length of the large intestine. At the end of the ninth month the meconium has reached the rectum.

3. **The condition of the gall-bladder.**—The gall-bladder begins to appear at the end of the fourth month, is distinct at the end of the fifth, contains insipid serous fluid at the end of the sixth, and bile at the end of the seventh month.

4. **The position of the testicles.**—At the end of the sixth month these lie close to the kidneys, and at the end of the

[1] Tidy, ii., p. 59 (1 line = $\frac{1}{12}$th of an inch).

seventh have begun to descend towards the internal ring, which they reach at the end of the eighth month. At the end of the ninth month they have, as a rule, passed through the canal and are often found in the scrotum.

5. **Miscellaneous characters.**—At the end of the fifth month the germs of the permanent teeth are visible; at the end of the sixth month the cerebral hemispheres cover the cerebellum. At the end of the seventh or eighth month the cerebral convolutions are apparent.

The chief of the above-mentioned characters will be found tabulated in the Appendix (*Tables XIX. and XX.*).

Question VII.—How far may the paternity of a child be inferred from its resemblance or non-resemblance to its alleged parents?

Undoubtedly, peculiarities of the parents are frequently transmitted to their offspring, *e.g.*: the general characters of the features, the colour of the skin, certain deformities, tendency to disease, tricks of manner, character of the voice, colour of the hair, &c., &c. Peculiarities in the parents are, however, not necessarily transmitted to their children, and, as before pointed out, a peculiarity may be subject to atavism, and miss one generation, appearing in the next. Further, the children of a woman by a second husband may resemble the first husband, not the second. More weight, therefore, is to be attached to the presence of hereditary peculiarities as affirmative evidence, than to their absence as negative evidence, of paternity. Other things being equal, the more close the resemblance, the stronger the evidence of paternity.

Question VIII.—Has this woman ever been delivered of a viable child?

The signs of recent delivery may be present and supply an affirmative answer. These signs will be discussed under Infanticide (see *post*, p. 369). Again the signs of virginity may be present. The presence of these, especially of an intact hymen, is a strongly-negative indication. An intact hymen may be taken as positive proof that the woman has never been delivered of a mature or nearly mature child. Obviously, however, no conclusion can be drawn from the absence of the signs of virginity.

If the signs of recent delivery and the signs of virginity are both absent, the chief indications available are—

(1) **The presence or absence of the lineæ albicantes and the condition of the breasts.**—The presence of the lineæ albicantes may, however, be accounted for by causes other than

delivery, *e.g.*, ovarian tumours, or ascites; and they may be absent in women who have been even more than once delivered.[1] Enlargement of the breasts also, may be the result of causes other than pregnancy.

(2) **The condition of the posterior commissure.**—This, if ruptured, strongly indicates a previous delivery. If intact, the indication is strong that the woman has never been delivered of a child, and still more strong that she has never been delivered of a mature child.[2]

(3) **The condition of the uterus.**—After delivery, the uterus does not wholly return to its original condition. The chief changes observable are as follows:—

(*a*) Its cavity becomes larger. According to Dr. Barnes,[3] the vertical diameter of the cavity is, in virgins 1·80, in women 2·20, and in mothers 2·44 inches; and the transverse diameter of the cavity, in virgins ·60, in women 1·08, and in mothers 1·24 inches.

(*b*) Its walls become thicker and its weight greater. According to Dr. Barnes, its weight, in girls at the age of puberty, is 360 to 1000 grains, whilst in women who have borne children its weight ranges from 1200 to 1800 grains. In advanced life, however (and in exceptional cases, in adult life after delivery), the uterus undergoes atrophy, and in old women its weight may become reduced to 100 to 200 grains.

On the whole, although the conditions of the uterus may afford strong indication of a previous delivery, no absolutely certain conclusion can be drawn from its state.

It may further be remarked that the question, Has this woman ever been delivered of a child? may also arise in defamation cases and in cases of disputed identity. In a recent trial for murder (*R.* v. *Wainwright, Case* C 34) this question arose with reference to the identity of the remains discovered and alleged to be those of a certain female who was missing. From the opinion expressed by Dr. Meadows in this case, it would appear that in the absence both of the signs of recent delivery, and of those of virginity, no certain answer can be given to this question.[4]

[1] Taylor, ii., p. 162.
[2] Tidy, ii., p. 138.
[3] Diseases of Women, p. 32.
[4] Taylor's Manual, p. 496.

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CHAPTER XXX.

INFANTICIDE.

HOMICIDE is destroying the life of a human being. Popularly the term "infanticide" is used to denote homicide where the human being killed is a newly-born infant. The law, however, draws no such distinction; in law, infanticide is homicide, and the provisions of the law which apply to homicide apply equally to infanticide. But although the law draws no distinction between infanticide and homicide, the subject of infanticide requires special consideration on account of, 1. **The frequency with which cases occur,** and 2. **The special nature of the medico-legal questions which arise.**

1. The frequency with which cases occur.

Infanticide is common in almost all countries, the motive being generally to get rid of an illegitimate child, or less commonly, to get rid of a child the parents are too poor to support. **In India two forms of infanticide** may be said to exist, viz.: (1) **Infanticide irrespective of the sex** of the child, and (2) **Infanticide of female children. As regards the first** of these forms of infanticide, the motives leading to it in India are similar to those which lead to it in other countries. Its frequency in India is however specially affected by certain social customs; viz.: (*a'*) **Early marriage,** which tends to diminish the frequency of the crime; and, (*b'*) **Prohibition,** especially among higher caste Hindoos, of **widow remarriage,** which tends to increase its frequency. As a consequence, therefore, while in European countries the accused is most frequently an unmarried female, in India the accused is very frequently a Hindoo widow **The second form of infanticide** may be said to be special to the East. In India the motives leading to it are: (*a*) Family pride among certain divisions of the warrior (Kshatri) caste, notably the Rajpoots and Thakoors, and consequent fear that a husband of suitable rank and position may not be found for the girl; (*b*) the extravagant expenditure entailed by custom on the parents at the marriage of a daughter, and (*c*) the disgrace which by social custom is attached to the father of a girl who attains puberty unmarried. Notwithstanding the fact that in India, owing to the

exertions of the British Government, this second form of infanticide had been rendered much less prevalent, it was considered necessary in 1870 to pass a special Act for its repression,[1] and even now the crime is far from rare. Some idea of the extent to which it was practised may be formed from the facts which came out in the course of an inquiry ordered by Government previous to the passing of the Act. It was found, for example, that in many villages of the Benares district, there were no girls at all. In Mynpoory again, out of thirty villages, in eleven there were no girls; and in the whole thirty, only 37 girls to 329 boys. Again in the N.W. Provinces, in seven villages inhabited by Rajpoots, there were 104 boys to one girl, and in nine other villages 71 boys to seven girls.[2] In Kathiawar and Kutch also the practice largely prevailed; in the latter province in 1840 "there were only 335 females to 4912 males of pure Jadeja (Rajpoot) blood."[3] Further it was shown that where measures for the repression of the crime had been adopted, the result was to greatly increase the number of female children. In Mynpoory for example, the number of Rajpoot girls rose in thirteen years from nil to 250, and in the Agra district the number of girls was doubled in a few years.

2. The special nature of the medico-legal questions which arise.

It has already been stated that the legal term "homicide" means the destroying of a human being. **According to the criminal law of England,** an infant is not considered a human being until the moment it is completely born; *i.e.*, completely and wholly external to the mother, irrespective of whether or no it be still attached to the mother by the umbilical cord. Hence, according to English law, the destruction of an infant before its complete birth has taken place, is not homicide.

According to the law of India, however, so far as homicide is concerned, a child is "in being" from the moment "any part of that child has been brought forth, though the child may not have breathed or been completely born."[4] Hence, according to Indian

[1] Act VIII. of 1870. The chief provisions of this Act are as follows:—Power is given to the Local Governments to apply the Act to any district or class, and thereafter (subject to confirmation by the Government of India) to make rules applicable to such district or class: (1) for the registration of births, marriages, and deaths; (2) for the regulation and limitation of marriage expenses; and (3) for the establishment of punitive police posts.

[2] Chevers, Med. Jurisp., p. 755.

[3] These figures in 1873 had risen to 4272 females to 8371 males (Cooke on the 'Repression of Female Infanticide in Bombay,' 1875).

[4] "The causing of the death of a child in the mother's womb is not homicide. But it may amount to culpable homicide to cause the death of a living child if any part of that child has been brought forth, though the child may not have breathed or been completely born" (Indian Penal Code, sect. 299, expl. 3).

law, killing an infant before any part of it is born is not homicide.

Further, sect. 315 of the Indian Penal Code states: "Whoever before the birth of any child does any act with the intention of thereby preventing that child from being born alive, or causing it to die after its birth, and does by such act prevent that child from being born alive, or causes it to die after its birth, shall, if such act be not caused in good faith for the purpose of saving the life of the mother, be punished with imprisonment of either description for a term which may extend to ten years, or with fine, or with both." Hence, according to this section, if the result of an act of the nature described therein, is to cause a child to die after its complete birth, the doing of the act is not punishable as culpable homicide. This is not so in England. By English law, the doing before the birth of a child of a felonious act, the result of which is to cause the child to die after its complete birth, is murder.

In cases of alleged infanticide therefore one of the questions arising is: Did the child survive its birth, partial birth, or complete birth, as the case may be? Usually the only data available on which an answer to this question can be founded, are the post-mortem appearances presented by the body of the infant. Hence arises the special question: What are the post-mortem appearances which indicate that an infant survived its birth? Again in cases of alleged homicide (infanticide included) expert evidence is usually required as to the cause of death. In infanticide cases, this question also involves the consideration of special points, *e.g.*, the causes connected with delivery which lead to the death of infants. Obviously also in a case of alleged infanticide, another special question which may arise is: Does this woman show signs of having been delivered (or recently delivered) of a child?

Hence the chief medico-legal questions which arise in cases of alleged infanticide, are: I. Did this child live after its birth?—This question, for the purposes of English criminal law, must be read as if the latter part of it stood, "after its complete birth," while for the purposes of Indian criminal law it must be read as if the latter part of it stood "after any part of it was born." **II. What was the cause of the child's death? and III. Does this woman exhibit signs of having been delivered (or recently delivered) of a child?**

Question I.—Did this child live after its birth?

It may first be remarked that in cases of alleged infanticide, it has always been the practice of the English Courts to require, in order to establish the fact of live birth, much stronger evidence than they will accept as proof of the same fact in civil cases. Next, it may be pointed out that in an infanticide case, it is

possible that the fact that the child lived after its birth, may be capable of being established by the evidence of ordinary witnesses, *e.g.*, of individuals who saw the child move, or heard it cry. In regard to crying as a proof of live birth, it must be noted that it is possible that a child may be heard to cry before birth, and while its head is still in the uterus (*vagitus uterinus*), or in the vagina (*vagitus vaginalis*). Several authentic cases of vagitus uterinus (Dr. Ogston has collected nine [1]) have been recorded (see *Case* C 35), and several others of vagitus vaginalis. In all the authentic cases of vagitus uterinus or vagitus vaginalis, which have been reported, a passage by which air could reach the mouth of the child was provided, by the introduction of the hand or instruments into the uterus or vagina. Although therefore it must be admitted that a child may be heard to cry before any part of it has been born, there is no doubt but that such cases are extremely rare, and have only been known to occur under the special circumstances stated above. Obviously, also, a child may be heard to cry after the birth of the head and before complete birth; the question, however, whether or no the crying took place after partial, but before complete birth, would not be material in a case of alleged infanticide in India, although it might be so in an English case.

As a rule, however, in cases of alleged infanticide, the only evidence of live birth available, is the opinion of an expert founded on post-mortem examination of the body of the infant. In giving such an opinion, the following points must be considered: 1. What is the degree of maturity of the child? 2. Does it show signs of having breathed? 3. Does it show any signs of live birth other than such as are directly due to the establishment of respiration? 4. Does it show signs of having been born dead?

Question I.—a. What is the degree of maturity of the child?

In order to establish the fact that infanticide has been committed, the law (both of England and of India) requires it to be proved that the child was born alive; not that it was born "viable" or capable of living and being reared. The degree of maturity of the child, however, is a factor which must be taken into account in framing an answer to the question: Did this child live after its birth? because the less the degree of maturity, the less the probability of live birth. Indeed in the case of a child born before the 120th day of intra-uterine life the possibility of live birth may be altogether excluded. (For the characters whereby the degree of maturity can be ascertained, see *ante*, p. 346, and *Appendix, Tables XVIII. to XX.*)

[1] Lectures, p. 247 *et seq.*

Question I.—b. Does this child show signs of having breathed?

Where respiration has been fully established certain changes will be found to have taken place in the lungs, viz.: (1) They alter in appearance and feel; (2) they increase in weight, and (3) their specific gravity is lowered.

(1) **Alteration in appearance, &c.**—The following table shows the changes produced.

Before Respiration.	After Respiration.
Dark red in colour.	**Bright red** or pink.
Collapsed, occupy only the upper part of the chest, leaving the pericardium exposed.	**Expanded** and nearly cover the pericardium.
Do not crepitate when handled or cut, and exude little blood on section.	**Crepitate** when handled or cut, and exude frothy blood freely on section.
No inflated air vesicles visible, but possibly bubbles of gas due to **putrefaction** present on surface of the lung. These are:—	**Inflated air vesicles** visible on surface of lung. These are:—
(1) **Large and not uniform** in size.	(1) **Small and nearly uniform** in size.
(2) **Not arranged in groups.**	(2) **Arranged in groups.**
(3) **Project considerably** from the surface of the lung, and	(3) **Project only slightly**, or not at all; and
(4) The gas in them **can be pushed** readily **from place to place.**	(4) **Cannot be pushed** from place to place.

(2) **They increase in weight**, owing to the increased amount of blood they contain. Hence two tests for establishment of respiration have been proposed: (*a*) **The absolute weight of the lungs** (Schmidt's test), and (*b*) **The ratio borne by the weight** of the lungs to the weight of the body (Ploucquet's test). **As regards the first of these tests**, Guy (from over 400 cases) gives the following as the average weight of the lungs in mature children: Before respiration, 874 grains; after respiration, 1072 grains. Individual cases, however, depart so greatly from these averages as to make this test most untrustworthy. Thus, in nine of Guy's cases, the weight of the lungs of children that had breathed was below 874 grains (the average in stillborn children), and in four of the nine below 600 grains. Again, Ogston gives a case where after respiration the lungs weighed only 420 grains. Further, in

three of Guy's cases the weights of the lungs of stillborn children were as follows: 1054, 1480, and 1950 grains, and in two of Ogston's cases, 1180 and 1315 grains. **As regards the second test: Ploucquet** (from three cases only) asserted that the ratio of the weight of the lungs to the body in mature children was, before respiration 1 to 70, and after respiration 1 to 35. Extended observations have, however, shown the average ratios before and after respiration to be much closer to one another than this. Thus Guy (from over 400 cases) found them to be respectively 1 to 57 and 1 to 38; Casper (from about eighty cases) 1 to 61 and 1 to 59; and Ogston (from sixty-six cases) 1 to 56·4, and 1 to 53·6. Here also individual cases depart so greatly from the average as to make the test untrustworthy. Thus, in individual cases, the ratio in stillborn children has been found 1 to 21, 1 to 24, and 1 to 37, and, in seven out of sixty-three cases of children born alive, Casper found the ratio to be between 1 to 74 and 1 to 99.

(3) **Their specific gravity is lowered.**—In the fœtal condition and before distension with air, the lungs are heavier than water. After distension with air they become lighter than water.[1] Hence if a portion of an undistended lung be thrown into water it sinks, while a portion of a distended lung floats. On this is founded the Hydrostatic test.

The Hydrostatic Test.

Procedure.—The test should be conducted as follows. The water employed should be of a specific gravity, as near 1000 as possible, *e.g.*, rain-water, or nearly pure water at 60° Fahrenheit.[2] With this a vessel, at least twelve inches deep and eight inches in diameter, should be nearly filled. The lungs with the heart attached having been removed from the body, are to be placed in the vessel, and it is to be noted whether they float or sink. Next each lung detached from the heart, is to be separately and similarly tried; then each lung is to be cut into about twelve pieces and each piece also tried. Lastly, it is to be noted whether the pieces, if they float, continue to float, after firm but moderate pressure has been applied to them. The pressure may be applied as follows: the pieces of lung having been wrapped in a cloth are placed on a hard floor or a board, and covered with another board. On this upper board the examiner should stand, avoiding any jerking movement.

Objections.—To the general proposition upon which this

[1] Taylor found the sp. gr. of undistended lungs to be 1·04 to 1·05, and the sp. gr. of the lungs of an infant that had breathed to be ·94.

[2] Rise of temperature lowers the sp. gr. of water; dissolved solid matters raise it. If the water is nearly pure, and its temperature is about 80° Fahr., about three ounces of common salt dissolved in five gallons of the water will restore its sp. gr. to about 1000. For water at 90° Fahr. the quantity required is about five ounces of salt to five gallons of water.

hydrostatic test may be said to be founded,—viz. that all portions of lung which have been distended with air float in water, while all portions which have not been so distended sink,—**certain objections have been raised.**

a. **It has been objected that undistended portions of lung may float in water,** owing to the presence in the intercellular tissue of (1) **Air-emphysema,** or (2) **Gases generated by putrefaction.** This undoubtedly may occur. But air or gases present in the intercellular tissue of a portion of a lung, may be expelled therefrom by moderate pressure. On the other hand, moderate pressure on a fair-sized piece of a distended lung, fails to expel any notable quantity of the air contained in its air-vesicles. Hence, if such a piece of a lung continues to float after moderate pressure has been applied to it, we may infer that the flotation is due to distension of its air-vesicles, and not to emphysema or putrefaction. When, however, the lungs are in an advanced state of putrefaction, it is possible that moderate pressure may not expel the whole of the gases present in the intercellular tissue; or may break up the lung tissue and cause expulsion of air from its air-vesicles. Hence it is advisable to refrain from drawing any inference from the floating or sinking of portions of the lungs, when these are in a highly putrid condition. *In situ*, however, the lungs putrefy comparatively slowly, and hence, unless general putrefaction of the body is far advanced, their condition is not likely to be such as to make it unsafe to draw decided inferences from the results of the hydrostatic test.

b. **It has also been objected that distended portions of the lungs may sink in water owing to disease,** *e.g.*, congenital tumours, œdema, congestion, hepatization, &c.[1] This is also true. Hence, therefore, if post-mortem examination shows the existence of very extensive disease of the lungs, we ought logically to refrain from drawing the inference that no portion of either lung has been distended with air, because all portions sink in water. Practically, however, as (1) cases of such very extensive disease of the lungs are extremely rare in newly-born infants, and (2) no harm results in criminal cases if the inference drawn is, that the lungs have not been distended; the objection that distended portions of the lungs may sink owing to disease, may be disregarded.

Inferences.—Given, then, that the following conditions are complied with: (*a'*) That the lungs are **not in an advanced state of putrefaction**; (*b'*) That **each lung is cut into** say twelve tolerably equal sized **pieces,** and (*c'*) That **firm but moderate pressure** has been applied to each piece; we may safely conclude that each piece that **sinks has not been distended** with air, and that each piece that **floats has been distended** with air.

[1] See *post*, Quest. II., Disease, p. 363.

Hydrostatic test as evidence of Respiration.

Suppose now, conducting the test as directed, we come to the conclusion (each piece sinking), **that no portion of either lung has been distended** with air, we may practically conclude that **respiration has not taken place** because: (1) Although infants have been known to live for several hours after birth without any portion of their lungs having become distended with air, such cases are extremely rare: and (2) In criminal cases, no harm results from drawing the inference that respiration has not taken place.

Suppose, however, conducting the test as directed, we come to the conclusion that some or all portions of the lungs have been distended with air, what inference may we then draw as regards respiration? Here it must be pointed out that distension with air may be due either to (*a'*) **artificial inflation,** or (*b'*) **natural respiration,** and that it is only when the first of these two causes of distension has been excluded, that we may with safety draw the conclusion that the distension observed is the result of natural respiration.

Artificial inflation is, however, **contra-indicated:** (*a'*) **If every portion of either lung is distended,** because it is extremely difficult even by skilled manipulation to effect complete distension of both lungs *in situ;* so difficult, in fact, that Ogston and others entirely deny the possibility of effecting it. (*b'*) **If the lungs on section exude much frothy blood,** and (*c'*) (but this is less reliable) **if the stomach and intestines are free from air.**

Of course, also, **the circumstances of the case may be, and in infanticide cases generally are,** such as to **exclude artificial inflation,** or at any rate skilled artificial inflation; and without skilled artificial inflation, it is extremely unlikely that any extensive distension of the lungs can be produced.

Hence, then, if attending to the precautions specified, we come to the conclusion (every piece floating), that **every portion** of the lungs has been **distended** with air, we practically, in cases of infanticide, may safely infer that **respiration has taken place.**

If, however, we come to the conclusion (some pieces floating and others sinking), that **portions** of the lungs **only** have been **distended** with air; the inference is, that **either artificial inflation** has been effected, or **natural respiration** has taken place. The question to which of these causes the distension is due, cannot be decided by the hydrostatic test alone, and in many cases also cannot be decided without taking into account the circumstances of the case.

It may be here remarked that the existence in the otherwise distended lungs, of portions in an undistended condition, has

been noticed in children in whom natural respiration has been established, and has been described under the name of atelectasis pulmonum.

Other evidences of respiration.—If the changes in the lungs indicate that respiration has taken place, confirmatory evidence of this indication may be afforded by the condition of the anterior chest-wall, and condition of the diaphragm. After respiration has been established, the former becomes more arched, and the upper surface of the latter becomes less convex and lies lower. Finding the upper surface of the diaphragm at a level corresponding to between the fifth and sixth ribs, may be regarded as confirmatory evidence of the establishment of respiration.[1]

Respiration as evidence of live Birth.

It must be pointed out that **the two questions,** Did this child **breathe?** and Did this child **live after its birth?** are not strictly concurrent, and that a negative or affirmative answer to the first, does not necessarily involve a similar answer to the second, because

(*a*) **A child may live after its birth without respiring,** or may respire so imperfectly that it may be impossible by post-mortem examination, to obtain satisfactory proof that respiration has taken place.

(*b*) **A child may respire before any part of it has been born.** That this is possible is shown by the fact that cases of vagitus uterinus and vagitus vaginalis have been recorded. Respiration before birth is, however, (1) only likely to occur in a case of face presentation, or under conditions similar to those present in the recorded cases of vagitus uterinus or vaginalis; (2) extremely rare; and (3) not likely to be anything more than imperfect, and not likely, therefore, to cause full distension of every part of the lungs.

These two cases excepted, it is obvious that in a criminal case in India, proof that respiration has taken place *de facto*, amounts to proof of live birth, and *vice versâ*. In criminal cases **in England a third** possibility must be excepted, viz.:

(*c*) **A child may respire after partial and before complete birth.** The possibility of the occurrence of this is beyond doubt. Whether, however, this has or has not occurred, cannot possibly be decided by post-mortem examination.

[1] The position of the diaphragm should be ascertained from below *before* the thorax is opened (see *Appendix XXIX., II.* 1 C.)

Question I.—c. Does this child show any signs of live birth, other than such as are directly due to the establishment of respiration?

The most important signs of live birth coming under the above description are:

(*a*) **Presence of food, e.g., milk, or of drugs, in the stomach.** This affords conclusive evidence of live birth.

(*b*) **Complete absence of meconium from the intestines.** In exceptional cases, the meconium is completely expelled before birth; but as a rule, its complete expulsion is not effected until some hours after birth. Hence complete absence of the meconium from the intestines, affords strong but not conclusive evidence of live birth.

(*c*) **Exfoliation of the scarf-skin.** This generally commences about the first day after birth, but sometimes not till later, and may not be completed for a month or more. It is difficult, however, sometimes to distinguish this vital change from peeling of the cuticle resulting from intra-uterine maceration.

(*d*) **Changes in and about the umbilical cord.**—These are: (1) Obliteration of its vessels (the arteries first), commencing about twenty-four hours after birth, and, according to Billard, taking place by concentric thickening. (2) Formation (generally about the third day) of a ring of inflammatory redness round the insertion of the cord, accompanied by thickening, and often by a slight purulent discharge. This ring of inflammatory redness must not be confounded with a narrower red line round the insertion of the cord, often present at birth. (3) Falling off of the cord occurring about the fifth day (in exceptional cases, as early as the second, or as late as the tenth); and cicatrization of the umbilicus, generally complete about the tenth to the twelfth day. Shrinking and withering of the cord commences soon after birth, but not being a vital change, is not a sign of live birth.

(*e*) **Closure of the special channels of fœtal circulation.**—(1) The internal portions of the umbilical arteries (hypogastric arteries); the internal portion of the umbilical vein, and its continuation the ductus venosus. The concentric thickening of these commencing at the umbilicus (see above) continues; at the end of two days, the arteries are contracted for the greater portion of their length, and by the end of the third day the contraction has nearly reached their termination in the iliacs. The vein and ductus venosus contract more slowly, showing only slight contraction for the first three days, which becomes more marked on the fourth day, and is, with few exceptions, complete on the fifth (Guy). (2) The ductus arteriosus. This begins to contract (at the aortic end first) as soon as respiration is established.

The contraction extends throughout the whole length usually during the first day. In a week, the channel becomes narrowed to the size of a crow-quill, and it is usually completely closed by the tenth day. (3) The foramen ovale usually closes between the second and the tenth day. **In exceptional cases,** closure before birth has been recorded in the case of the foramen ovale; and within, at any rate, ten minutes of birth, in the case of the ductus arteriosus. No case of closure before birth of the ductus venosus is on record. The foramen ovale sometimes does not close till the end of the second year. Sometimes the foramen ovale, or the ductus arteriosus, remains patent throughout life, giving rise to cyanosis.

(*f*) **Other signs of live birth** which have been proposed are: (1) Emptiness of the urinary bladder; this is wholly unreliable, and (2) Presence of air in the cavity of the tympanum, replacing the gelatinous matter with which this cavity before birth is filled. This indicates that respiration has taken place. As, however, the replacement may not occur for five weeks (Tidy), absence of air from the cavity of the tympanum is wholly unreliable as a sign of still-birth, or of death soon after birth.

Certain of the foregoing signs of live birth may be utilized for the purpose of determining how long a child has survived its birth (see *Tables XXI. and XXII., Appendix*, p. 509).

Question I.—d. Does this child show signs of having been born dead?

The body may show signs of intra-uterine maceration. This is readily distinguished from ordinary putrefaction. In intra-uterine maceration (1) The odour exhaled by the body differs markedly from the odour of ordinary putrefaction. (2) The skin is coppery red, or flesh-coloured, not green. (3) The bones are more or less separated, and the body is flaccid; the head, thorax, and abdomen flattening out when the body is placed on a level surface. It must be recollected, however, (*a*) That when death has occurred shortly before delivery, signs indicating intra-uterine maceration will be absent; and (*b*) that after delivery, ordinary putrefaction may supervene and mask the appearances resulting from intra-uterine maceration. Hence, therefore, while the presence of distinct signs of intra-uterine maceration amounts to positive proof of still-birth, no inference can be drawn from the absence of such signs.

CHAPTER XXXI.

INFANTICIDE—*continued.*

Question II.—What was the cause of the child's death?

I.e. was it due to (a) natural causes, (b) violence, or (c) neglect or omission.

Question II.—a, Death from natural causes.

CHILDREN, owing to natural causes, are frequently born dead. **Still-birth is more frequent** (*a*) in first than in subsequent pregnancies, (*b*) in male than in female children, and (*c*) among illegitimate than among legitimate children. Statistics show the proportion of still to live births to be about 5¼ per cent. of the total number of births, and to be (*a*) about 9 per cent. in first as compared with about 3·2 per cent. in other pregnancies; (*b*) about 5·8 per cent. in male as compared with about 4·7 per cent. in female children; and (*c*) about twice as great among illegitimate as among legitimate children. Death from natural causes may be due to:—

1. **Immaturity and consequent debility.**—When a child is born alive, and dies after its birth, solely in consequence of debility arising from its want of maturity, the question arises, Was the premature delivery, as a result of which the child was born immature, criminally induced or not? If criminally induced, an offence has obviously been committed. By the law of India, however, this offence is not punishable as culpable homicide.[1] On the other hand, according to the law of England, provided, of course, the child lived after its complete birth, the offence which has been committed is murder.[2]

2. **Debility not due to immaturity.**—A mature infant free from disease, may die from debility, and consequent inability to continue breathing. In such a case the lungs will most probably be found, at any rate in parts, imperfectly distended, and portions may be found in a condition of atelectasis (see p. 359).

3. **Disease.**—This may be general disease, *e.g.*, smallpox,

[1] See Penal Code, sect. 315, p. 358.
[2] See case of *R.* v. *West*, Taylor, ii., p. 313.

syphilis, or cancer; or **local disease.** If the latter, the seat of the disease may be the lungs, brain, or heart.

(1) **Congenital disease of the lungs.**—This, according to Guy, may be (*a*) hepatization—red or grey—from pneumonia before birth; (*b*) pulmonary apoplexy; (*c*) tubercle; (*d*) œdema; or (*e*) Devergie's œdema lardaciforme.

(2) **Congenital disease of the brain** and cord may be (*a*) morbid softening, "but it must be borne in mind that the brain of the fœtus is naturally soft and vascular;"[1] (*b*) effusion of blood (apoplexy) into the substance, cavities, or on to the surface of the brain; or (*d*) effusion of other fluids, *e.g.*, serum or pus.

(3) **Congenital disease of the heart** or large vessels is rare in infancy. Tidy also mentions uric acid infarction, *i.e.*, blocking of the kidney tubes with uric acid or urates, as a cause of infant mortality.

Obviously post-mortem evidence of the existence of disease does not necessarily prove death therefrom: much must depend on the extent of the diseased condition.

4. Malformation.—Death may be due to a congenital malformation, *e.g.*, of the heart or large vessels; or of the alimentary canal, such as an imperforate gullet or anus. No amount of malformation or monstrosity justifies the destruction of the infant.

5. Hæmorrhage from apertures of the body may cause death, *e.g.*, from the genitals of a female infant, or from the rectum. Two cases of death from the last-mentioned cause are recorded by Casper.

6. Protracted or complex labour frequently results in the death of the infant. The immediate cause of death may be—

(1) **Accidental violence** to the body of the child (see Violence).

(2) **Exhaustion from protracted labour.**—Death from this cause is frequently accompanied by marks of violence on the body of the child, especially on the head, but may not be so accompanied. Protracted labour is more common in first than in subsequent deliveries; and the greater the disproportion between the size of the child and that of the mother's pelvis, the more likely is labour to be protracted. Male children are generally larger than female children: hence more male than female children die during delivery.

(3) **Fœtal asphyxia.**—Asphyxia may occur before birth owing to premature separation of the placenta, or be due to (*a*) **the death of the mother,** or (*b*) **obstruction to the flow of blood through the cord.** With reference to (*a*), Tidy remarks that there is more chance of saving the child when the mother has died suddenly, than when her death has occurred slowly. Garezky concludes that in most cases infants are more or less asphyxiated after the first minute, but that they may be extracted alive in a

[1] Guy, For. Med. p. 116.

more or less asphyxiated condition, up to twenty-six minutes after the death of the mother. Harris considers that a child may live still longer (one to two hours). From Tidy's summary of 379 cases of post-mortem Cæsarean section, it appears that in 81·3 per cent. of the cases the children were dead when extracted, and in 9 per cent. distinctly alive, but of these only one-seventh lived for any length of time. **As regards** (*b*), **the obstruction** to the flow of blood may be due to **pressure on the cord** from abnormal presentation, *e.g.*, foot or breech, or from prolapse of the cord. Scanzoni gives nearly 55 per cent. as the mortality in cases of prolapse of the cord. Again, the obstruction may be due to the **accidental formation of a tightly drawn knot** on the cord: two knots even have been found; and lastly, the obstruction may arise from spontaneous rupture of the cord during delivery.

Question II.—b, Death from Violence.

Death from violence may be the result of **accident**, or the violence may have been inflicted **intentionally**; if the latter, under Indian law, it will be a material question whether or no death resulted from an act done before the birth of the child (see *ante*, p. 353). Again, death from violence may be due to **mechanical violence, or to poison**; in the former case, the mode of death may be **asphyxia**, from suffocation, drowning, or strangulation; or **coma**, from head injury: or **syncope**.

1. Asphyxia from suffocation.—**Accidental** suffocation may occur in many ways, *e.g.*, from the head being born enveloped in the membranes; from pressure of the child's face against soft bedding; from the child being overlaid by some one in the same bed; from some spasmodic retraction of the tongue; or from accidental entry of particles of food into the air-passages. **Intentional suffocation** is a frequent mode of infanticide. The following are the more commonly adopted methods: (1) drawing the membranes tightly over the child's head; (2) closure of the mouth and nostrils by the hand or a cloth; (3) stuffing mud or rags into the mouth, or plugging the fauces with a piece of cotton-wool, sometimes, in India, smeared with mustard oil; (4) rolling the tongue back into the throat; (5) burying the child's face in bran, or in mud, cow-dung, or other matters. Any matters found in the throat of the infant, should be carefully examined and preserved.

2. Asphyxia from drowning.—**Accidental drowning** (or suffocation) may occur from the infant falling into a privy or cesspool, owing to the mother being suddenly delivered while in the act of defæcation: this may occur even in primiparæ (see *post*, *Coma*). Finding the cord torn across,[1] not cut, supports the

[1] Usually about two inches from the navel (Guy). See also p. 369.

supposition of the occurrence of such an accident. Accidental drowning may also occur from the infant, at the time of delivery, falling face downwards into the mother's discharges. **Intentional drowning** is sometimes resorted to. In some parts of India, immersion of the child's face in milk is a common method of infanticide.

3. Asphyxia from strangulation.—**Accidental strangulation** may occur from the funis becoming tightly coiled round the neck of the child, or, in rare cases, by the spasmodic contraction of the os round the neck of the child. **Intentional strangulation,** by the fingers, funis, or other ligature, is a frequent form of infanticide. As before mentioned (see *Hanging and Strangulation*), if the ligature employed is a soft one, *e.g.*, the funis, no mark may be left on the neck. Casper points out that natural folds on the skin of the neck, especially of fat infants, somewhat resemble marks caused by a ligature. Close examination and dissection of such marks, however, will show no extravasation and no condensation of tissue.

If the **post-mortem appearances** show that death has been due to **asphyxia,** much will depend on the absence or presence of **marks indicating** that the asphyxia has been **due to violence. If all such marks are absent,** death may have been the result either of accident or intention; it is not possible from the post-mortem examination to say which. **If such marks are present,** much will depend on what they are. Some of themselves strongly indicate intention, *e.g.*, finger-marks on the throat, or rags, &c., firmly impacted in the fauces. Others are consistent with either accident or intention, the probabilities being more in favour of intention than accident, if the amount of violence has been great. A torn cord (see *post*, p. 369) supports the supposition of accident.

4. Coma.—**Accidental death** from coma, due to head injury, may occur before labour, during labour, or after delivery.

(1) **Before labour.**—In very exceptional cases fracture of the skull of the fœtus in utero has been caused by violence applied to the body of the mother. Thus, Ogston (p. 269) mentions a case where a fracture of the right parietal bone, one inch in length, resulted from the mother, during a fit of hysteria, falling out of bed four weeks before delivery (see also *Case* C 36). Great violence may, however, be applied to the body of the mother without injury to the fœtus.

(2) **During labour.**—Pressure on the head from expulsive efforts during delivery may cause death from coma, either without or with fracture of the skull. The first, and much the most common case (without fracture), is the most frequent cause of death during delivery. In such cases, inside the cranium will be found congestion of the brain and its membranes, and in rare cases extravasation of blood. Outside the cranium a sero-sanguinolent tumour (*caput succedaneum* or *cephalhæmatoma*)

frequently forms. Extravasations of blood under the scalp, due to pressure on the head, are, it should be noted, larger in size and more irregular in outline than the punctiform ecchymoses which occur under the scalp in cases of suffocation (see *ante*, p. 89). Cases of fracture of the skull by expulsive efforts during labour are recorded, but such fractures are rarely more than mere fissures, generally of the parietal, less frequently of the frontal, bone. Defective ossification of the cranial bones, obviously favours the production of such fractures. **Injury to the head may be the result of efforts to effect delivery.**—If the result of the mother's own efforts, the skull is not likely to be more than slightly fractured. Extensive injury may obviously be produced by the use of instruments; and considerable injury may be produced without the use of instruments, by ignorant efforts to aid delivery (see *Case* C 38)

(3) **After delivery.**—If a woman is delivered in an erect position and the child falls on a hard floor, fatal injury to the head, with or without fracture to the skull, may occur. The possibility of this is shown (*a*) by the fact that several cases of this accident have been recorded (see Taylor, ii., 399); and (*b*) by Casper's experiments on the bodies of infants, which conclusively prove that a fall from a height of 30 inches suffices to cause fracture of the skull. In every one of Casper's experiments, twenty-four in number, fracture was actually produced: confined, however, to the temporal bones in twenty-two out of the twenty-four cases. As before noted, sudden delivery, leading possibly to such an accident, may occur even in primiparæ (see *Case* C 37). Finding the cord torn across (see p. 369) supports the supposition that such an accident has occurred. The length of the cord is important in such cases, and should, if possible, be ascertained, fracture being more likely to occur if the cord be short. The average length of the cord is eighteen to twenty inches; it, however, has been found as short as four to six, and as long as fifty-four inches. **Intentional violence** to the head is a common mode of infanticide. In homicidal cases the amount of violence employed is usually very great.

On the whole, therefore, if death has resulted from injury to the head, and there is **no fracture or punctured wound** of the skull, it is almost certain that death was due to **accident. Slight fractures** of the skull bones are also perfectly consistent with **accident. Extensive injury** to the head is strongly in favour of **homicide,** especially if accompanied by a cut cord, and unaccompanied by severe injury to the mother.

5. **Syncope or shock.**—This may occur from (1) hæmorrhage from the divided cord; (2) external wounds; (3) fractures or other internal injuries.

(1) **Hæmorrhage from the divided cord** is more likely to occur (*a*) when it has been cut across with a sharp instrument,

than when it has been divided with a blunt one or torn asunder; (*b*) when it has been divided close to the umbilicus; (*c*) when it has been divided almost immediately after birth; and (*d*) when it is thick and gelatinous. **Fatal hæmorrhage from the cord** may possibly but not necessarily occur. (*a*) **If the cord has not been tied,** and may occur even if the cord has been torn and not cut. Spontaneous rupture of the cord may occur during delivery; the usual mode of death from this accident is, however, asphyxia, not syncope. (*b*) **From the ligature not being tied sufficiently tight,** or from its slipping: or (*c*) **From the navel after separation** of the cord (secondary hæmorrhage).

Obviously, **when the cause of death is primary hæmorrhage** from the divided cord, it is important to note (*a*) whether or no the free end of the cord appears to have been cut, and (*b*) whether or no a mark of a ligature is present.

(2) **External wounds.**—Death from syncope or shock, the result of external wounds, is usually homicidal. Fatal external wounds may, however, be the result of accident, *e.g.*, from broken utensils, or the result of an obstetric operation. The nature of the injury may show whether it is the result of accident or design. Fatal injury, it may be noted, may be caused by wounds which leave hardly any external mark, *e.g.*, the thrusting of a needle through the fontanelles, the temples, under the upper eyelid and through the orbital plate of the frontal bone, or through the back of the neck into the spinal cord or brain, or the thrusting of a sharp instrument down the throat or up the rectum. Cases have occurred of the employment of each of these methods. Such injuries obviously strongly indicate homicide; but Ogston,[1] it should be mentioned, records a case of death from accidental thrusting of a small pin through the anterior fontanelle.

(3) **Fractures or other internal injuries.**—Just as fractures of the skull may occur before, during, or after delivery; so fractures of other bones, or dislocations of joints, may similarly occur. Cases even are recorded of women, who have met with no accident during pregnancy, being after an easy labour, delivered of an infant with more than one of its long bones fractured or dislocated. Fractures or dislocations are not likely to prove immediately fatal, unless the neck be the seat of the injury. Twisting the neck is a frequently employed method of infanticide. Very great force is required to effect this, and hence death from this cause strongly indicates homicide. Harvey,[2] however, records a case where a woman in her unaided efforts to effect her own delivery, the case being one of breech presentation, employed so great an amount of force, apparently without homicidal intent, as to tear the body of the child completely away from its head.

6. Infanticide by Poison.—In India poisoning by opium is

[1] Lectures, p. 268. [2] Beng. Med. Leg. Report, p. 314.

said to be a commonly employed method of infanticide; and it is alleged that in some cases a peculiar mode of administration is adopted, viz., smearing the mother's nipples with the drug. Harvey, however, doubts whether wilful poisoning by opium is as common as is popularly believed to be the case. Opium is largely used (in India as crude opium, and in Europe in the form of syrups containing opium) by women of the lower classes to keep young children quiet. Hence accidental cases of the poisoning of young children by opium are of common occurrence. Other poisons, said to be used in India for the purposes of infanticide, are tobacco and madar (Calotropis procera), which see. Taylor mentions cases of intentional poisoning of young children by arsenic, sulphuric acid, and phosphorus scraped from the heads of lucifer matches. Arsenic is also sometimes similarly employed in India. Accidental poisoning of young childen by arsenic has been known to occur, as, for example, in a recent case in England, where a number were poisoned by the external application of arsenious oxide, introduced as an adulterant into "violet powder." It must not be forgotten that in new-born infants, post-mortem appearances simulating those of irritant poisoning, are sometimes met with as the result of disease, *e.g.*, injection of the mucous membrane of the œsophagus, and ulceration of that of the stomach and intestines. Such appearances have been met with in the bodies of plump and fat children.[1]

Question II.—c, Death from Neglect or Omission.

Sect. 32 of the Indian Penal Code states: "In every part of this code, except where a contrary intention appears from the context, words which refer to acts done extend also to illegal omissions." Death from neglect or omission may be accidental or intentional, and causing death by an intentional and illegal omission may or may not amount to murder. **The principal forms of neglect or omission** likely to cause death are—

1. **Omission to provide assistance during labour.**—This may result in the death of the infant from suffocation, head injury from a fall, hæmorrhage from a ruptured cord, &c., &c. (See *ante, Death from violence.*) Two questions which may arise in such cases are: (1) Is it possible for a pregnant woman to remain ignorant of her state up to the time of her delivery? That this, in exceptional cases, is possible, has already been pointed out (see *Pregnancy*, p. 333); and (2) Would a newly-delivered woman be capable of the exertion necessary to save the life of her child? As regards this second question, it may be remarked (*a*) that in rare cases women have been delivered during profound natural sleep (see *Case* C 39); and (*b*) that in some cases women have been known to go through a considerable

[1] Ogston's Lectures, p. 272.

amount of exertion immediately after delivery (see *Cases* C 37 and 40). As a rule, however, a newly-delivered woman is capable of but little exertion (see, however, *post*, p. 370).

2. **Omission to tie the cord after dividing it.**—If a woman has been delivered without assistance, proof that the cord has been cut, not torn, indicates that ability existed after delivery for a certain amount of exertion. This, taken with the other circumstances of the case, might support the supposition that the omission to tie was intentional. Previous to examining the cut end of the cord, this, if dry, should be softened in warm water. A clean smooth edge indicates that the cord has been cut; a ragged edge may be the result of division with a blunt instrument or rupture.

3. **Omission to supply the infant with food or to protect its body against cold** may cause its death. In the first case absence of all signs of the presence of food in the alimentary canal may, it is possible, indicate the cause of death. In the second case there may be no distinctive post-mortem appearances present. Sometimes this omission takes the form of "Abandonment" of the infant. This is an offence, even if death does not result, for by sect. 317 of the Indian Penal Code, "Whoever being the father or mother of a child under the age of twelve years, or having the care of such child, shall expose or leave such child in any place with the intention of wholly abandoning such child, shall be punished with imprisonment of either description for a term which may extend to seven years, or with fine, or with both." For a curious legal point arising under this section, see *Case* C 41, *R.* v. *Beejoo*

Lastly, it may be pointed out that by section 318 of the Indian Penal Code it is an offence "by secretly burying or otherwise disposing of the dead body of a child, whether such child die before, or after, or during its birth," to intentionally endeavour to conceal "the birth of such child." Women are frequently convicted under this section when the evidence fails to support a graver charge.

Question III.—Does this woman exhibit signs of having been delivered (or recently delivered) of a child?

The signs of previous delivery, in cases where the signs of recent delivery are absent, have already been discussed (see p. 349). Question III. may, therefore, now be limited to recent delivery. **On examination during life of a woman who has recently been delivered of a mature, or nearly mature, child, the following signs will usually be found:—**

1. **A general appearance of indisposition.**—This, however, may be present in women, who have not been recently delivered,

as the result of any severe illness. Again, this sign may be absent in women who have been recently delivered. Some women, especially those accustomed to labour, appear to be constitutionally but little affected by delivery, and are capable immediately afterwards of resuming their work or undergoing severe exertion. Chevers,[1] on the authority of Ward, states that poor women in the northern parts of Bengal are known to attend to the business of their families the day after delivery; and that sometimes a mother is delivered while at work in a field, carries home the child, and returns there to work the next day. For an instance of very considerable exertion directly after delivery, see *Case* C 40.

2. **A swollen, contused, or even lacerated condition of the organs of generation.**—A laceration of the fourchette is usually found after delivery in primiparæ. The os uteri also may be found lacerated, and is dilated and soft. The uterus is enlarged; and, Ogston[2] remarks, may for the first two or three days be found to undergo alternate contraction and relaxation, under pressure of the hand applied to the abdomen. **The abdominal parietes** are relaxed, the lineæ albicantes apparent, and a dark line is seen extending from the pubes to the navel.

3. **The breasts,** as in advanced pregnancy, are full and prominent, and the nipples surrounded by well-marked areolæ. Milk will be found exuding from the nipples.

4. **The Lochial discharge.**—The presence of this discharge is the most characteristic sign of recent delivery. It is at first coloured with blood, afterwards becomes brown or green, and has a peculiar odour. The discharge may become almost wholly suppressed about the third or fourth day under the influence of the milk fever, returning when this has subsided. It usually lasts a week to a fortnight, but may continue longer. Ogston[3] states that in some instances the lochia have been known not to appear at all.

Many of these signs may be present as the result, not of delivery, but of uterine or ovarian **disease.** No conclusion can, therefore, be safely drawn, unless all, or nearly all, the signs of recent delivery be present. As a rule, the signs of recent delivery cease to be distinguishable after the eighth to the tenth day; and the stronger the woman, and the less severe the labour, the more likely are they to disappear rapidly. The earlier the period of gestation also, at which delivery has taken place, the less marked will be these signs and the more quickly will they disappear. For further signs of recent delivery, ascertainable on post-mortem examination, see *Causing miscarriage*, p. 380.

[1] Page 744. [2] Lectures, p. 155. [3] *Ibid.*, p. 158.

CHAPTER XXXII.

CAUSING MISCARRIAGE

THE term "miscarriage," as used in law, includes both abortion and premature labour. Medical writers, however, use the term "premature labour" to denote premature expulsion of a child that has attained viability, and the terms "abortion" or "miscarriage" to signify expulsion of an ovum or fœtus at any earlier period. Miscarriage may be—

I. **Accidental,** *i.e.*, the result of natural or accidental causes.
II. **Justifiable,** *i.e.*, the result of a lawful act; or
III. **Criminal,** *i.e.*, the result of an unlawful act.

I. ACCIDENTAL MISCARRIAGE.

This frequently occurs, and is more common in the earlier than in the later stages of pregnancy. Whitehead, from observation of 2000 pregnancies, estimates that one in seven end in abortion. Dr. Robert Barnes divides the causes of accidental or natural miscarriage into (A.) Maternal and (B.) Fœtal causes, and classifies them thus:—

(A.) Maternal Causes.

1. **Poisons circulating in the mother's blood:** (*a*) Introduced from without, as fevers, syphilis, various gases, lead, copper, &c. (*b*) Products of morbid action, as jaundice, albuminuria, carbonic acid from asphyxia, and in the moribund.
2. **Diseases degrading the mother's blood:** Anæmia, obstinate vomiting, over-lactation.
3. **Diseases disturbing the circulation dynamically** (mechanically), as liver, heart, and lung disease.
4. **Causes acting through the nervous system:** (*a*) certain nervous diseases, as chorea, &c.; (*b*) mental shock; (*c*) diversion or exhaustion of nerve force, as from obstinate vomiting.
5. **Local diseases:** (*a*) Uterine diseases, as fibroid tumours, inflammation, hypertrophy, &c., of the uterine mucous membrane; (*b*) mechanical anomalies, as retroversion, pressure of tumours external to uterus, &c.
6. **Climacteric abortion.**
7. **Abortion artificially induced.**

(B.) Fœtal Causes.

1. **Diseases of the membranes of the ovum.**—As fatty degeneration, hydatidiform degeneration, inflammation, congestion, apoplexy, and fibrous deposits.

2. **Diseases of the embryo itself.**—Malformation, inflammation of serous membranes, diseases of nervous system, diseases of kidneys, liver, &c., and mechanical, as from torsion of the cord.

Common causes of accidental miscarriage are syphilis, mental shock, and accidental violence. In some women miscarriage results from the slightest exciting cause. Others having once miscarried, miscarry in subsequent pregnancies apparently without any exciting cause. Others, again, seem "proof against the most severe physical injuries and suffering" (see *Cases* C 42 and 43) "and the most violent mental excitement."[1]

II. JUSTIFIABLE MISCARRIAGE.

Sect. 312 of the Indian Penal Code, defining the offence of causing miscarriage, formally excepts as not criminal, miscarriage caused "in good faith and for the purpose of saving the life of the woman." The law of England does not formally define under what circumstances it is lawful to cause miscarriage. Usually justifiable miscarriage takes the form of "artificial induction of premature labour," *i.e.*, the operation is deferred until the child has attained viability, so that, if possible, its life as well as that of the mother may be saved. So long, however, as the operation is undertaken for the purpose of saving the life of the mother, miscarriage may be legally caused at any period of pregnancy. For the purpose of saving the mother's life it may be necessary to cause premature expulsion of the contents of the pregnant uterus:—

1. **In cases where from pelvic distortion** the antero-posterior diameter of the pelvis (normally $4\frac{1}{4}$ inches at the brim and $4\frac{2}{3}$ inches in the cavity) is reduced below, or to, $3\frac{1}{4}$ inches.

2. **In cases of obstruction** caused by the presence of tumours, or contractions of the soft parts arising from cicatrices, of such a nature as to prevent the passage of a mature child.

3. **In cases where during gestation the mother's life is endangered** by obstinate vomiting, hæmorrhage from placenta prævia, convulsions, or serious cardiac, or pulmonary, or other disease.

Dr. Meadows and others advise the artificial induction of premature labour in "cases in which there is evidence that on

[1] Tidy, ii., p. 156.

several previous occasions the death of the fœtus occurred at a given time suddenly. Here," writes Meadows, "the operation would be resorted to prior to the period in question with the view of preventing its recurrence"[1] To cause miscarriage under these circumstances is by the law of India not justifiable, unless there is reason to believe that the child's death will endanger the life of the mother.

III. CRIMINAL MISCARRIAGE.

Criminal abortion, or miscarriage, is common in many countries, the object being to get rid of the product of illicit intercourse. In India the custom of preventing the re-marriage of widows tends directly to increase the prevalence of the offence. In India, in fact, in by far the great majority of cases of this offence, the female who has miscarried is a Hindoo widow (see *Cases* C 24 and 25). This, however, is not invariably the case (see *Case* C 26).

The sections of the Indian Penal Code relating to the offence of causing miscarriage are as follows:—

312. "Whoever voluntarily causes a woman with child to miscarry, shall, if such miscarriage be not caused in good faith for the purpose of saving the life of the woman, be punished with imprisonment of either description for a term which may extend to three years, or with fine, or with both; and if the woman be quick with child, shall be punished with imprisonment of either description for a term which may extend to seven years, and shall also be liable to fine."

"*Explanation.*—A woman who causes herself to miscarry is within the meaning of this section."

313. "Whoever commits the offence defined in the last preceding section without the consent of the woman, whether the woman is quick with child or not, shall be punished with transportation for life, or with imprisonment of either description which may extend to ten years, and shall also be liable to fine."

314. "Whoever with intent to cause the miscarriage of a woman with child, does any act which causes the death of such woman, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine; and if the act is done without the consent of the woman, shall be punished either with transportation for life, or with the punishment above mentioned."

"*Explanation.*—It is not essential to this offence that the offender should know that the act is likely to cause death."

Two other sections of the code refer to results which may arise to the child from the doing of certain acts before its birth, viz., sect. 315 (see p. 353); and

[1] 'Manual of Midwifery,' p. 234.

316. "Whoever does any act under such circumstances that, if he thereby caused death, he would be guilty of culpable homicide, and does by such act cause the death of a quick unborn child, shall be punished with imprisonment of either description for a term which may extend to ten years, and shall also be liable to fine."

Attempts to cause miscarriage may be dealt with either by the application of the provisions of sect. 511 of the Penal Code (see p. 3) to sects. 312 or 313; or if the attempt has been made by the administration of an "unwholesome drug or other thing" the case may be dealt with under sect. 328 (see p. 111).

Hence by the law of India to voluntarily cause or attempt to cause "miscarriage," except in good faith for the purpose of saving the life of the woman, is an offence. Further, supposing it to be proved that such an offence has been committed, the following **additional questions** are, owing to the wording of the above-quoted sections, liable to arise:—(1) **Was the woman pregnant?** Proof of pregnancy is required to secure a conviction for causing miscarriage, but not to secure conviction for an attempt. (2) Was the woman **quick with child?** (3) **Was the miscarriage caused,** or the attempt to cause it made, **without the consent** of the woman? (4) **Did the woman's death result** from the miscarriage or the attempt to cause it? And (5) In certain cases (see sects. 315 and 316) **did the death of the child result** from an act done before its birth?

In England, causing miscarriage is punishable under sects. 58 & 59 of 24 & 25 Vict. c. 100, as amended by 27 & 28 Vict. c. 47 (see *Appendix XXIII.*). It may be noted that under these sections, (1) the question of pregnancy only arises when a woman is accused of doing an act with intent to procure her own miscarriage; (2) that the question of quickening does not arise at all; (3) that these sections do not, like those of the Indian Code, expressly provide that the absence of the woman's consent aggravates the offence. Further, in England, if the death of the woman or the child results, the ordinary law of homicide applies.

In investigating a case of alleged criminal causing of miscarriage, examination may have to be made of: 1. **The substances** alleged to have been expelled. 2. **The means** alleged to have been used; and 3. **The female** alleged to have miscarried.

1. Examination of the substances alleged to have been expelled from the uterus.

(*a'*) **These may contain no ovum or embryo;** *e.g.*, they may be blood clots, a fibroid tumour, a dysmenorrhœal false membrane, a mole, or hydatids. In India, in such a case, the accused can only be convicted of an attempt to cause miscarriage. In England, except the accused be the female alleged to have

miscarried, the nature of the substances expelled from the uterus is immaterial.

(*b'*) **They may contain an ovum, embryo, or immature fœtus.**—As by the law of India causing miscarriage is punishable with greater severity if the woman be quick with child, it is important to determine the uterine age of an immature fœtus found in the matters expelled. For the characters of the fœtus at various periods of gestation, see p. 347, and *Appendix XVIII. to XX.* In criminal miscarriage, it may be noted, the usual period selected is during the fifth or sixth month. Chevers, however, points out that women in India "not infrequently induce premature confinement when they have nearly advanced to their full period."[1] In giving, from examination of the fœtus, an opinion as to whether quickening has occurred, it must be borne in mind that quickening does not take place at any fixed period (see *Pregnancy*, p. 337). After ascertaining the probable uterine age of the fœtus, the question of the cause and time of its death must next be considered. By the law of England, the fact that the death of the child resulted from criminal causing of miscarriage, only affects the gravity of the offence committed in one case, viz., where the child survives its complete birth. In such a case the offence committed is murder according to English law. This is not so in India. In India, however, in certain cases (see Penal Code, sects. 315 and 316) the fact that the death of the child resulted renders the offender liable to enhanced punishment.

(*c'*) **What has been expelled may be a mature child.**—In such a case it is of course possible that miscarriage has not occurred at all. When this is suspected, signs indicative of maturity should be carefully looked for. One of the most important of these, only available however if the child be dead, is the presence in the lower epiphysis of the femur, of a point of ossification more than three-quarters of a line in width (see p. 348). Of course, in all cases where the degree of maturity of the child indicates that it might possibly have been born alive, the questions whether or no it survived its birth, and what was the cause of its death, must be inquired into, as in a case of alleged infanticide.

2. Examination of the means alleged to have been used.

Miscarriage may have been caused, or an attempt to cause it made, by (1.) General violence; (2.) Local violence; or (3.) Administration of drugs.

[1] Chevers, p. 735.

(1.) General Violence.

Blood-letting has, in some countries, a popular repute as a means of causing miscarriage. Bleeding from the foot, application of leeches to the anus or to the vulva, have all been resorted to. Blood-letting, however, usually fails, and the same may be said of the use of very hot or very cold baths. **Violent exercise,** such as hard riding; severe jolting, as driving over a rough road; **violent shocks,** as from blows or falls; and tight **compression** of the abdomen, may all cause abortion. Often severe general mechanical violence altogether fails (see *Cases* C 42 and 43). Sometimes mechanical violence is combined with the administration of drugs (see *Case* C 44).

(2.) Local violence (including local applications).

Rupturing or separating the membranes, with or without dilation of the os; the insertion of a foreign body into the uterus; or the use of the vaginal douche; are the means of inducing premature labour commonly employed by medical practitioners. Similar means are often resorted to by unskilled persons in criminal attempts, and often fatal injury to the mother is the result. **In India** a common method employed is the introduction into the vagina or uterus, of either a twig of some irritant plant, or a thin piece of stick armed with some irritant or reputed abortifacient preparation. The plants, twigs of which are most commonly used, are **Plumbago rosea** and zeylanica (Chitra), and less frequently **Nerium odorum** (oleander), **Cerbera Thevetia** (yellow oleander), and **Euphorbium Tirucalli** (milk bush). The twigs of these plants, previous to introduction, are often smeared with asafœtida (*vern.* Hing). When a piece of stick armed with an irritant or other preparation is employed, the stick is commonly wrapped round at one end or for the greater portion of its length with cotton, and to this the preparation is applied. More or less common ingredients of the preparations employed for arming such sticks are, besides matters derived from the plants just named, the juice of **Calotropis procera** (Madar), the milky juice of various **Euphorbias,** bruised **marking-nuts, arsenious oxide, orpiment,** and **red lead.** This last (red lead), very commonly in Bombay, forms one of the ingredients of the composition employed. Sometimes a mass of irritant paste is simply thrust into the upper part of the vagina. Not infrequently the twig or stick employed passes wholly into the uterus, causing fatal inflammation, and after death is found lying wholly or partly within the cavity of the emptied or unemptied uterus, perhaps transfixing its walls. **Perforation of the uterine wall** by such sticks or twigs may occur from force used in introducing them,

or from subsequent contraction of the uterus upon them. I recollect being present in Bombay at a post-mortem examination on an abortion case, in which two thin pieces of stick were found lying side by side in the uterus across the fundus, both transfixing the uterine wall on either side. From their position, these sticks must have been (*a*) retained by the uterus after expulsion of its contents, and (*b*) forced through the uterine wall by the contractions of the uterus. Introduction of irritant twigs, or of sticks armed as described above, appears to be the method usually resorted to in India by practised abortionists, and very often proves successful. It should be noted that irritant substances are sometimes introduced into the vagina as a mode of torture. Dr. W. Gray met with a case in which a man placed three marking-nuts in his wife's vagina, as a punishment for infidelity.[1]

(3.) Administration of Drugs.

The substances popularly believed to possess abortifacient properties, may conveniently be arranged in five classes, viz. (*a*) Ecbolics; (*b*) Reputed Emmenagogues; (*c*) Purgatives; (*d*) Irritants, and (*e*) Other substances.

(*a*) **Ecbolics,** *i.e.*, substances which stimulate the contraction of the muscular fibres of the uterus. The only undoubtedly ecbolic drug known is **Ergot.** Administration of this, after the uterus has begun to contract, nearly always increases the force and frequency of its contractions. When, however, contractions of the uterus have not commenced, administration of ergot may or may not excite their commencement. Apparently, the less advanced the pregnancy, the more likely is it to fail. Hence, when given with criminal intent, as is frequently the case in England, it often fails to cause abortion. Ergot has been stated to act injuriously on the child. Dr. U. West,[2] however, records that out of one hundred and seventy-two labours in which he gave ergot, only five still-births resulted, or considerably less than the usual percentage of still to live births.[3] **Borax** has been stated to possess ecbolic action, but this is extremely doubtful. Harvey[4] mentions a case in which borax (Sohagar, Tankanakhara) is said to have been one of the ingredients of a preparation taken by a woman convicted of causing her own miscarriage. **Cotton root bark** is said to act on the uterus like ergot, and has been used as a substitute for it.[5]

(*b*) **Reputed Emmenagogues**—*i.e.* substances believed to promote the menstrual flow. The principal substance of this class used criminally as an abortifacient, is **Savin** (Juniperus

[1] Bombay Chem. Analyser's Rep., 1874–75.
[2] Taylor, ii., p. 192.
[3] Five and a quarter per cent. (see p. 362).
[4] Bengal Report, p. 301.
[5] Lauder Brunton's Pharmacology, p. 788.

Sabina). This is frequently employed in England, both in the form of the powdered leaves (or a decoction made from them), and in the form of oil of savin. It often occasions abortion, but often fails. When given in large doses for the purpose of procuring abortion, it acts as a powerful irritant poison, and has in several cases caused death. The following reputed emmenagogue, poisonous plants, have also been criminally employed in Europe:—**Rue** (Ruta graveolens), **Yew** (Taxus baccata), **Tansy** (Tanacetum vulgare), and, in India, **Oleander** (Nerium odorum and Cerbera Thevetia). All these are powerful poisons: the first two have caused abortion, the others are not known to have any effect on the uterus. **Belladonna** applied locally as a suppository, or used as a vaginal injection, is stated to have caused abortion. Single cases of abortion following internal administration of **Actea racemosa** (black snake root, or cohosh), and **Digitalis**, the latter resulting fatally to the mother, have also been reported.

Less active or non-poisonous drugs of this class are:—**Pennyroyal** (Mentha Pulegium). This has been used in England for the purpose of procuring abortion. Most authorities consider it to be without action on the uterus, and many do not even consider it to be a noxious substance.[1] Tidy, however, doubts its absolute innocence.[2] **Papai seeds** (Carica Papaya), and **carrot seeds** (Daucus Carota, *vern.* Gajir-bij), are both popularly believed in India to be powerfully abortifacient. In regard to the first, Dymock [3] states that the general belief among all classes of women in Southern and Western India s, that if a pregnant woman partakes of them even in moderate quantity, abortion will be the probable result. As regards the second, numerous cases are recorded where carrot seeds have been given internally, their administration being followed by abortion. More precise information is much wanted as to the alleged abortifacient power of both these drugs.

(*c*) **Purgatives**, especially such as cause much straining, or act powerfully on the rectum, may, if given in large doses, bring on abortion. This effect is more likely to result in the advanced, than in the earlier stages of pregnancy. **Aloes**, in the form of **Hierapicra** (a mixture of powdered aloes and powdered canella bark), and **Pilacotia** (a mixture of aloes and colocynth) has frequently been used in England as an abortifacient. **Sulphate of potash** is said to be much employed for the same purpose in France. **In India**, in the Bombay Presidency, I have met with cases where various **Cucurbitaceous tubers**, viz., **Cucumis trigonus** (Karit), **Momordica Charantia** (Karela), and **Momordica Cymbalaria** (Kadavanchi), have been used, it is alleged, with success. Again, Taylor gives an English case where one hundred and twenty grains of **Colocynth** taken with intent to excite abortion, caused death.[4] **Elaterium, Croton oil**, and

[1] Taylor, ii., p. 185.
[2] Tidy, ii., p. 169.
[3] Mat. Medica of W. India, p. 295.
[4] On Poisons, p. 522.

Gamboge may also be classed as purgatives, likely, under certain circumstances, to cause abortion.

(*d*) **Irritants.**—Powerful irritants may, like purgatives, cause abortion, owing to the uterus participating in the irritant action set up in the system. Obviously, if given to an extent which renders abortion probable, the death of the mother is likely to result. Among the **mineral irritants** which have been used, may be mentioned: (1) **Arsenic**; this is sometimes employed in India; it has, in more than one case, caused death without producing abortion (see *Cases* C 23 and 24). (2) **Iron**; the sulphate, and the tincture of the perchloride, have been used in England: neither of these preparations appear to possess any specific abortifacient power. (3) **Mercury**; this has been used in England in the metallic form, and as calomel, but without success. Chevers mentions an Indian case where mercuric sulphide formed one of the ingredients of a powder given with intent to cause abortion. (4) **Copper**; in another Indian case mentioned by Chevers, sulphate of copper formed one of the constituents of a similar powder. (5) **Quicklime**; this is also mentioned by Chevers as used for the purpose of procuring abortion. (6) **Iodide of potassium and Nitrate of potassium.** In one case each, the administration of these salts was followed by abortion; iodide of potassium is, however, frequently in the course of medical treatment, given to pregnant females, without ill effect. **Organic irritants.**—**Plumbago** (rosea and zeylanica), the juice of various **Euphorbias**, and the juice of the **Calotropis procera**, are all in more or less common use in India for internal administration as abortifacients. Chevers also mentions as similarly used—**Black Pepper**, unripe **Pine apple**, the bark of **Moringa pterygosperma** (horse-radish tree), and **blistering flies**; and Gray,[1] in a case of abortion followed by death from irritant poisoning, identified the drug used as the leaves of **Lasiosiphon speciosus** (N. O. Thymelaceæ, *vern.* Rametha; see p. 212). **Cantharides**, Tidy states, has in some cases caused abortion, and in others failed, even in doses of one drachm of the powder.

(*e*) **Other substances.**—Numerous other substances, none of which so far as known possess any specific abortifacient power, are mentioned by various writers as enjoying more or less popular repute as ecbolics. **In India**, the juice of **Bamboo leaves**; the fruit of **Randia dumetorum** (*Mainphal*, or *Gelaphal*), an emetic recommended as a substitute for ipecacuanha in dysentery; a decoction of **Cuscuta reflexa** (Akasweli, Ghagar bel); the seeds of **Celastrus paniculata** (Malkangni); and the seeds of **Anethum graveolens** or Sowa (Indian dill), have all been used. See also, under Poisons, Dolichandrone falcata, and Plumieria acutifolia. In Europe, squills, hellebore, and laburnum (see Poisons), have all three been employed as abortifacients. So also have the

[1] Bombay Analyser's Report, 1874–75.

following: sarsaparilla, Guinea pepper (grains of paradise), saffron, guaiacum, horehound, camomile, wormwood, mugwort, and juniper.

3. Examination of the female.

During life, traces left by the means employed may be found on the person of the female: *e.g.*, bruises on the abdomen, marks of injury on the genitals, or foreign bodies in the vagina. If miscarriage has actually been caused, the signs of recent delivery may be present. These obviously may be expected to be less marked, the earlier the period of gestation at which the miscarriage has taken place, and the longer the interval which has elapsed since it occurred. Harvey mentions a case where no signs of an alleged abortion at three months were present seven days after the event. On the other hand, the same author mentions a case where relaxation of the genitals was found six or seven days after abortion at two to two and a quarter months, and another, where in a woman æt. twenty-two, eight days after abortion at four months, the following signs were found: vagina slightly dilated, puerperal smell distinct, the uterus could be felt through the abdominal wall, and a little milk could be squeezed from the breasts. In other cases, Harvey states that signs sufficient to indicate abortion, were reported to be present a fortnight to a month after the occurrence.[1]

After Death.—Further traces left by the means employed may be found, *e.g.*, extravasation of blood underlying bruises, internal wounds, signs of irritation on the mucous membrane of the alimentary canal, presence of poisons, &c. In addition to the signs of recent delivery present during life, others become available, derived from examination of (1) the uterus, and (2) the ovaries.

1. **The Uterus.**—This may be found enlarged, the enlargement being greater the more advanced the period of gestation at which delivery took place, and the less the time which has elapsed since the event. Montgomery gives its dimensions a day or two after delivery at the full term, as 7 to 8 inches by 4 inches, and its weight as $1\frac{1}{2}$ lbs. Fourteen days after delivery at the full term, it does not exceed 5 inches in length, and weighs about $\frac{3}{4}$ lb. If delivery has taken place at five months, the uterus, according to the same authority, will be found immediately afterwards to measure $5\frac{5}{8}$ by $3\frac{3}{4}$ inches; and fourteen days afterwards, $4\frac{1}{2}$ by $2\frac{3}{4}$ inches. On internal examination within a few hours of delivery at the full term, coagula, or fluid tinged with blood, will be found in the cavity. At the seat of attachment of the placenta, the substance of the organ will be found exposed, showing large valvular openings. The inner surface is extremely

[1] Bengal Med. Leg. Rep., 1870–72, p. 297.

dark, almost black, in colour, and portions of the decidua, intermixed with flakes of lymph, adhere to it. These appearances also are less marked the earlier the period of gestation, and the longer the time which has elapsed since expulsion of the uterine contents. As already pointed out, twigs of irritant plants, or pieces of stick, may, in abortion cases, be found in the cavity of the uterus, or transfixing its walls.

2. **The Ovaries.**—Ordinarily at each menstruation an ovum escapes from the ovary, leaving behind it a cicatrix called a **corpus luteum.** As a rule, this cicatrix undergoes a peculiar development during pregnancy; but does not undergo such development, if the escape of the ovum is not followed by pregnancy. Hence corpora lutea are distinguished as true and false, meaning by a "**true corpus luteum,**" the corpus luteum of pregnancy, and by a "**false corpus luteum,**" the corpus luteum of the unimpregnated female. In some exceptional cases, the development of the cicatrix and its conversion into a body not distinguishable from a true corpus luteum, has been found to occur in the unimpregnated female; and *vice versâ*, in other exceptional cases, no such developed cicatrix has been found in a pregnant female. According to Tidy and others, true and false corpora lutea, three weeks after the escape of the ovum, present the same characters, viz., cavities three-quarters of an inch in diameter, containing a reddish-coloured central clot, and having a pale convoluted wall. About one month after the escape of the ovum, the convoluted wall in both becomes bright yellow, but the false corpus luteum has diminished, while the true corpus luteum has increased in size. In the false corpus luteum, the diminution in size goes on rapidly: at two months it has become "an insignificant cicatrix," and at six months it has entirely disappeared. On the other hand, the true corpus luteum at two months is seven-eighths of an inch in diameter, its convoluted wall still retains its bright yellow colour, but "the central clot is perfectly decolorised." At six months it is still of the same size; the convoluted wall, although paler, is still yellow, and the central clot "appears fibrinous." At nine months, its diameter has become reduced to half an inch, the yellow colour of the convoluted wall has disappeared, and the central clot "is converted into a radiating cicatrix."[1] Hence, then, the presence in one of the ovaries, of a body possessing distinctly the characters of a true corpus luteum, is strong, but not conclusive evidence, of pregnancy or delivery. The absence of such a body is similarly strong, but not conclusive, evidence to the contrary.

[1] Tidy, ii., p. 124.

Post-mortem delivery.

In examining the dead body of a female alleged to have miscarried, the possibility of the occurrence of this accident must not be forgotten. Post-mortem delivery, owing to the pressure of gases evolved during putrefaction, may occur after death at any period of gestation. It may or may not be accompanied by inversion of the uterus. Inversion even of the non-gravid uterus may occur from the same cause. Harvey, summarizing the Bengal Medico-legal Reports for the three years ending 1872, mentions that nine or ten cases of post-mortem delivery, and several of post-mortem inversion of the non-gravid uterus, were reported during this period. For a typical case of post-mortem delivery, see *Case* C 45.

CHAPTER XXXIII.

LIFE ASSURANCE.

WHEN an individual assures his life, he in effect enters into an agreement with a company to pay them, each year he lives, a certain fixed sum, or "**premium.**" In return, the company issue a "**Policy of Assurance,**"[1] or undertaking to pay a certain fixed sum on the death of the assured, whenever this may happen.[1] For a given sum, payable at death, the yearly premium to be paid by the assured must obviously be more or less, according to his "**expectation of life,**" *i.e.*, according to the number of years he may reasonably be expected to live. An individual's expectation of life depends (1) on his age, and (2) on his freedom or otherwise from any special influence tending to shorten life. If no such special influence exists, the individual's expectation is said to be **normal.** Hence arises the general question, on the answer to which the ordinary scale of premium rates of assurance companies must obviously be based, viz.:—I. What is the normal expectation of life at various ages?

But an individual seeking to assure, may be subject to some special influence tending to reduce his expectation of life, and hence two other questions arise, viz.:—II. What are the special influences which tend to shorten life, and to what extent do they do so? and III. How is the existence, in any given case, of influences tending to shorten life ascertained?

QUESTION I.—WHAT IS THE NORMAL EXPECTATION OF LIFE AT VARIOUS AGES?

By calculation from statistics of mortality, tables may be constructed, showing the expectation of life among individuals subject to the same conditions as those to whom the statistics refer. Table XXIV. (*Appendix*, p. 510) is an abstract of two of the most recently published tables of expectation of life in

[1] This is the simplest form of the agreement; variations, however, are frequently introduced. Thus the yearly premium may be made payable in monthly, quarterly, or half-yearly instalments; or the assurance may be effected for a term of years only, the liability of the company ceasing on the expiry of the stated term; or it may be arranged that the sum assured shall be paid on the assured attaining a certain age, or at his death, whichever may first happen.

England. On these, and similar tables, assurance companies base their ordinary English premium rates, *i.e.*, the rates they charge for assuring the lives of individuals residing in England, whose expectation of life is normal (see also Table XXV., *Appendix*, p. 511). Approximately, the expectation of life shown by these tables may—for ages between twenty-five and seventy-five—be arrived at by Willich's formula, viz., that the expectation at a given age is equal to two-thirds of the difference between eighty and the age: *e.g.*, at age thirty this formula gives, as the expectation of life, two-thirds of fifty ($=80-30$), or thirty-three and one-third years, a figure very nearly that shown by the tables. No tables have as yet been published showing the normal expectation of life among natives of India. The rates, however, at which one of the principal assurance companies in India assures healthy native lives, appear to correspond roughly to an expectation—between twenty and sixty—of two-fifths of the difference between ninety and the age: *e.g.*, at age thirty the expectation apparently calculated on is about twenty-four years, *i.e.*, two-fifths of the difference between thirty and ninety.

QUESTION II.—WHAT ARE THE SPECIAL INFLUENCES WHICH TEND TO SHORTEN LIFE, AND TO WHAT EXTENT DO THEY DO SO?

It is convenient, in the first place, to point out that when an individual, proposing to assure, is found to be subject to a special influence tending to shorten life, an assurance company may either refuse altogether to undertake the risk, or may agree to assure the life, charging an enhanced rate of premium as a compensation for the individual's diminished expectation. This enhanced rate may be charged in one or other of the following ways.

(1) According to a special table of rates fixed by the company for individuals subject to a particular influence: *e.g.*, residence in a tropical climate.

(2) The ordinary premium rate for an individual whose expectation of life is normal may be charged, plus a special additional rate, calculated either as a percentage on the sum assured, or on the ordinary premium. This is the method commonly adopted when the influence reducing expectation is the individual's occupation.

(3) By adding a certain number of years to the assured's age, and charging him, instead of the ordinary rate corresponding to his actual age, the ordinary rate for an individual so many years older. This is the plan generally followed when the influence reducing expectation is the existence of disease, or of a predisposition to disease. It should be noted that, when this method is adopted, the number of years to be added to the age must

always be greater than the number of years by which it is estimated that the individual's expectation is reduced. A little consideration will show that, where Willich's formula is applicable, the addition must, in round numbers, be one and a half times the reduction of expectation, *i.e.*, just so many years as will raise the assured's actual age to the age at which the reduced expectation exists.[1]

The special influences tending to shorten life may conveniently be considered under three heads, viz.:—1. External; 2. Congenital; and 3. Acquired influences.

1. External influences.

The chief external influences likely to reduce expectation of life are—1. Locality of residence; and 2. Occupation.

1. Residence in an unhealthy locality.—Practically, assurance companies may be said to recognise three classes of localities, viz. (1) specially unhealthy, (2) unhealthy, and (3) ordinary localities. The West coast of equatorial Africa is considered to belong to the first class, and companies will only assure lives resident there by special agreement and at a special rate. India and tropical countries generally are looked on as belonging to the second class. During time of residence in a locality of this second class, some companies charge, in addition to the ordinary premium, an extra rate, in some cases as much as 1½ to 2 per cent. per annum on the sum assured. Many companies, however, publish a special table of rates for residence in India and the tropics. The rates shown in those tables vary considerably: approximately the amount by which they exceed the corresponding ordinary or English rates, ranges from ¾ to 2¼ per cent. per annum on the sum assured.[2] In forming an estimate of an individual's expectation of life, it should not be forgotten that of a number of localities to which the same assurance office rate applies, some may be more unhealthy than others. Low-lying, marshy districts, for example, are more unhealthy than well-drained ones, and in England towns are, as a rule, less healthy than country districts. Thus Guy gives the expectation of life at thirty for the whole of England, as 34·1 years. The records of benefit societies in rural districts, however, show an expectation of 38·4 years at the same age; while in Liverpool and Glasgow the

[1] For more precise calculations a table of expectation of life must be employed thus:—Find the expectation corresponding to the individual's actual age; from this deduct the number of years by which it is estimated his expectation has become reduced; then find in the table the age corresponding to the reduced expectation; the difference between this and the actual age is of course the number of years to be added.

[2] In the Appendix will be found (Table XXVI.) an abstract of two tables of expectation of life in India, viz. (A) of a table compiled by J. Westland, Esq., Beng. C. S., from the experience of the Bengal U. C. S. Fam. Pension Fund (European and Eurasian lives), and (B) of a table compiled by A. F. Cox, Esq., Mad. C. S., from the combined mortality statistics of the Bengal, Madras, and Bombay Civil Services (European lives only). From these tables some approximation as to the extent by which the expectation of healthy Europeans is reduced by residence in India may be arrived at. The expectation shown by Table B, it will be observed, corresponds approximately to half the difference between the age and eighty-six (not two-thirds of the difference between the age and eighty).

expectation at thirty is respectively twenty-seven and twenty-five years.[1] In India, again, certain localities are well known to be more unhealthy than others.

2. Occupation.—The occupation of an individual may tend to reduce his expectation of life by exposing him to risk of (1) mechanical injury, (2) absorption of poison, or (3) contraction of disease, or of a habit tending to shorten life.

(1) **Mechanical Injury.**—The chief occupations exposing to this risk are as follows:—**Military and Naval service.** In war time extra rates of five to twenty guineas per cent. have been charged to officers actually engaged. During time of peace officers of the Navy are usually charged an extra rate of half a guinea per cent. within certain limits, and a special higher rate beyond.[2] In India an extra charge of about 1½ per cent. per annum (which covers war risk in India) is usually made for military employ. **Engine-drivers, sailors, and miners.** The usual extra charge for these occupations is 1 to 2 per cent. additional on the sum assured. Other occupations exposing to this risk, and for many of which extra rates are charged, are mining engineers and agents, makers of explosives, quarrymen and others using explosives, railway officials generally, policemen, firemen, gamekeepers, builders, plumbers, and glaziers, and all occupations involving constant contact with horses.

(2) **Absorption of poison.**—This risk attends the manufacture of chemicals generally, and specially to the manufacture of compounds of the more poisonous metals. Occupations involving constant contact with such metals or their compounds (*e.g.*, arsenic, mercury, lead, and copper), are also exposed to it. Again, occupations involving exposure to poisonous vapours: *e.g.*, phosphorus vapour, nitrous acid vapour, sewer gases, &c., involve this risk.

(3) **Contraction of disease, or of a habit tending to shorten life.**—Occupations exposing to risks of this kind are (*a'*) **very dusty occupations,** as grinders, millers, masons, and coal-miners; Sieveking remarks that few of the Sheffield steel grinders attain the age of thirty-five; (*b'*) **sedentary occupations,** especially if carried on in badly ventilated rooms; (*c'*) **occupations likely to lead to the acquirement of habits of intemperance,** *e.g.*, publicans and others engaged in the manufacture or sale of alcoholic liquors. Sieveking[3] points out that while between the ages of forty-five and fifty-five the general mortality for all England is 18 per 1000, the rate between the same ages in the case of inn and beershop keepers is 28 per 1000, and in butchers (probably from the same cause) 23 per 1000. There is some reason also to suppose that much railway travelling injuriously affects health, and hence that occupations involving this tend to shorten life. It may also be noted that, according to Guy, the average duration of life among members of the learned professions is seventy-six years and six months, or greater than is the case in any other occupation.

2. Congenital influences.

1. Liability to inherit disease.—The percentage of heredity of different diseases, *i.e.*, the percentage of cases in which hereditary transmission is traceable, is variously stated by different authorities. Much of this variation arises from difference in the fact accepted as showing hereditary transmission. Thus, if the only fact accepted as indicating this, is affection of the parents, a lower percentage of heredity will be found, than when affection

[1] Guy, quoted by Sieveking, 'Medical Adviser in Life Assurance,' p. 119.
[2] Sieveking, 'Medical Adviser in Life Assurance,' p. 121.
[3] *Op. cit.*, p. 62.

of the grandparents or any of their children is accepted. The following are the chief hereditary diseases, and the main facts derived from European experience, bearing on the question under consideration. Little or no information is available as regards hereditary transmission of disease among natives of India.

(1) **Pulmonary consumption.**—The percentage of heredity of this disease is variously stated at 25 to 60 per cent. Females appear to be somewhat more liable to inherit it than males, and the disease seems to be more liable to descend from mother than from father to child. Sieveking considers that where the personal condition of the individual is good, the death from consumption of one parent, or of two of the individual's brothers or sisters, should be met by an addition of seven to ten years to the age; and that if both parents have died of the disease, the life should either be rejected altogether, or twenty to twenty-five years added to the age.

(2) **Gout.**—Percentage of heredity equals about 50. Sieveking states that the usual practice is to add three years to the age for hereditary liability to gout, but considers this addition inadequate.

(3) **Cancer.**—Percentage of heredity, 8 to 33. Twice as many females as males die from this disease; hence the addition to age for hereditary liability to cancer, should be greater in the case of females.

(4) **Rheumatism.**—Percentage of heredity, about 30. Acute rheumatism, although not so likely to prove directly fatal as other hereditary diseases, may damage the heart and so impair expectation.

(5) **Insanity, and brain disease generally.**—Percentage of heredity of insanity, 25 to 60. Hereditary transmission of insanity appears to be more common in the upper than in the lower classes of society, and to take place more frequently from mother to daughter, than from father to son. Other brain diseases, *e.g.*, epilepsy, exhibit a marked tendency to hereditary transmission, and frequently brain disease in one form in the parents is transmitted in another form to the children. Contrary to a notion formerly entertained, insanity undoubtedly tends to shorten life.

(6) **Syphilis and scrofula.**—Hereditary transmission of these diseases is chiefly liable to affect the expectation of life of an adult indirectly, *e.g.*, by rendering him less able to resist an attack of serious disease.

2. Indirect hereditary influences.—Great disparity of age (and, according to some, near consanguinity) between the parents, or extreme youth of the mother, may exert an indirect injurious effect on an individual's expectation of life, by interfering with his development, or power of resisting attacks of disease. Under the head of indirect hereditary influence, the influence of sex on expectation may also be considered.

The general expectation of life among females is slightly greater than among males. Females, however, are subject to the special risk attendant on child-bearing. This risk does not attach to the pregnant condition, but to parturition, and attaches specially to a first delivery. Dr. Allen's statistics, collected from various sources, give as the proportion of deaths (from puerperal causes) to deliveries, one in sixty-two for primiparæ, and one in one hundred and twenty-four for multiparæ. Females, therefore, pregnant for the first time, Sieveking advises,[1] should

[1] *Op. cit.*, p. 75, "the older the primipara the heavier should be the rate."

be charged a special rate, and it is a question whether an extra rate should not also be charged to multiparæ. The risk to life attendant on parturition is greatly increased by insanitary surroundings: hence the advisability of charging an extra rate becomes greater, if the female about and after the time of her delivery is likely to be subject to such surroundings. Such extra rates may be remitted when parturition has taken place, or the period of child-bearing has come to an end. Frequent previous miscarriages often indicate a syphilitic taint, and justify an extra rate being charged, no matter what may be their cause.

3. Acquired influences.

1. **A previous attack of disease** may be deemed to have reduced expectation, if the disease is (1) serious in nature and likely to recur, *e.g.*, cancer, apoplexy, epilepsy, gout, &c.; or (2) likely to have impaired the functions of some important organ, *e.g.*, acute rheumatism, dysentery, sunstroke, &c.; or (3) one indicating serious constitutional taint, or impairment of function, *e.g.*, fistula, piles, &c.

In some cases the expectation may be deemed to be so greatly reduced as to render the life uninsurable. In other cases the reduction of expectation may be met by an addition to the age. No general rules can be laid down applicable to all cases as to the course which should be followed. Sieveking, however, states that a single well-marked attack of acute rheumatism, confining the individual to bed for six weeks or more, justifies an addition of seven to ten years to the age, and that the addition should be greater if there has been a recurrence of the disease. The same authority also considers the usual deduction of three years from expectation for an attack of gout too little.[1]

2. **Acquirement of a particular habit.**—The habit reducing expectation, most commonly coming under notice, is intemperance. Intemperate habits, according to Mr. Neison, reduce expectation so greatly as to bring it down (in the middle ages of life) to about 19½ minus one-fifth of the age; *e.g.*, at thirty to 19½ minus six, or 13½ years. Abuse of narcotics other than alcohol, *e.g.*, opium (which see), and habits other than over-indulgence in narcotics, may also reduce expectation.

3. **Existence of a morbid or abnormal condition,** such as:—

(1) **Blindness.**—Usually met by an addition of ten years to the age. (2) **Hernia.**—This, unless the individual agrees to wear a truss, renders his life uninsurable. (3) **Loss of a limb,** or malformation interfering with the power of locomotion. For loss of a leg three years is usually added to the age, but this Sieveking thinks is insufficient. (4) **Open ulcers.**—These must be healed before the life can be accepted. (5) **Deafness.**—No addition is usually made for this, although, like blindness, it undoubtedly exposes the individual to increased risk of accident. (6) **Presence of disease.**—See *post*, Examination of the proposed Assuree.

[1] *Op. cit.*, pp. 97, 143.

Finally, it should be pointed out that in the same case there may exist a combination of influences reducing expectation. The locality of residence, occupation, or habits of an individual may, for example, have the effect of augmenting the damage caused to his expectation of life by disease or a tendency thereto. Thus residence in a tropical climate may augment the damage to expectation resulting from certain diseases of the digestive organs. An occupation involving much exertion may augment the damage due to certain affections of the circulatory system; or one involving much anxiety of mind, the damage due to a tendency to brain disease. Intemperate habits again increase the damage due to disease generally, and specially damage resulting from affections of the nervous and digestive organs.

QUESTION III.—HOW IS THE EXISTENCE IN ANY GIVEN CASE OF INFLUENCES TENDING TO SHORTEN LIFE ASCERTAINED?

The usual method is as follows: The individual proposing to assure is—(1) Supplied by the insurance company with a series of printed questions, to which written answers are required. (2) Required to refer to two or more persons personally acquainted with him (one being his usual medical attendant), and to these "referees" of the proposed assuree a similar series of questions are addressed. (3) Examined by a medical man acting on behalf of the company, who also is usually supplied with a series of questions to be put to the proposed assuree; and (4) Required to sign a formal declaration to the effect that his statements are true, and are to be taken as forming the basis of his contract with the assurance company.

As regards (1), The object of the questions put to the proposed assuree is of course to ascertain whether or no he is subject to any hereditary or other influence tending to shorten life. In answering them (and generally) the assuree is bound to exercise the utmost good faith in the representations he makes to the assurers; failure in this respect on his part will, as a general rule, render the policy void. Further, save in very exceptional cases, misrepresentation or concealment of material facts will render the policy void. Hence arises a fourth question in regard to life assurance, which presently must be considered, viz.: Has there been misrepresentation or concealment of material facts?

As regards (2), Any person the assuree nominates as one of his "referees" may refuse to act in such capacity; but if he undertakes the duty, he is bound, like the assuree, to exercise the utmost good faith in discharging it, at the risk, should he fail in doing so, of rendering himself liable to an action should loss ensue. Hence "the usual medical attendant" of the pro-

posed assuree, if he accepts the duty of a referee, is bound to answer truly all questions put to him, and to disclose every material fact known to him; or, should he have no knowledge as to any particular fact, in regard to which information is required from him, to state so distinctly.

As regards (3), The examination of the proposed assuree should be thorough, and, in order that nothing may be omitted, should be conducted in regular order, something as follows.

Examination of the proposed Assuree.

General external examination.—This may (*a*) directly or (*b*) indirectly disclose the existence of a condition tending to shorten life. The chief conditions coming under head (*a*) are blindness, hernia, loss of a limb, or malformation interfering with the power of locomotion, open ulcers, and deafness (see *ante*, p. 388). Under head (*b*) may be mentioned (1) **Gait, manner, and general appearance.** This may indicate actual disease of the nerve centres or of a tendency thereto, or premature decay; the individual looking older than his age; or existence of a habit tending to shorten life, *e.g.*, intemperance. (2) **Skin disease**: a disease of this class may indicate a constitutional taint or intemperate habits. (3) **Weight**; this should be in fair correspondence with the height. From *Table XXVII., Appendix*, p. 512, it will be seen that roughly a male European, 5 feet 7 inches in height, should weigh about 150 lbs., and 5 lbs. more or less for every inch above or below this height. The proportion borne by the weight appears, in the case of natives of many districts of India, to be lower than among Europeans (see *Table II., Appendix*, p. 493). Very low or very high weight in proportion to the height, or marked recent gain or loss in weight, should be looked on with suspicion. A variation in weight of more than 20 per cent. from the standard, is regarded by most authorities as incompatible with normal health.

The various systems of the body should next be examined. It may be remarked that in **England** the percentage of deaths from diseases of the chief systems to total deaths, is about as follows:—Respiratory, 30 (one-third of these from phthisis); nervous, 13; circulatory, nearly 7; digestive, about 5½; and genito-urinary, about 2 per cent. **In India** a very large proportion of the total deaths (in the Bombay Presidency about 65 per cent.) are attributed to "fever." In ordinary years, "bowel complaints" come next, being credited with (in Bombay) about 8½ per cent. of the total deaths. In exceptional years, the deaths from cholera exceed those from bowel complaints. Thus, in 1877 (the famine year), in Madras,[1] 22 per cent., and, in Bombay, 9 per cent. of the total deaths were reported as from cholera.

[1] The total death-rate in Madras in 1877 was 53·2 per 1000.

Respiratory system.—Respiration should be quiet and easy, its ratio to the pulse 1 to 4 or 5, and not quicker than 20 per minute. The chest should expand in all directions, and there should—especially below the clavicles—be no flattening. Deep inspiration should cause no distress. There should be no lividity of the lips, or tips of the ears or fingers; and the individual ought to be able to count aloud rather slowly 1 to 20 or 30, without taking fresh breath. The circumference of the chest should be in fair correspondence with the height, and in suspicious cases the "vital capacity" (*i.e.*, the volume of air expelled after the deepest possible inspiration by the deepest possible expiration) should be ascertained. For average chest measurement and vital capacity corresponding to different heights, see *Table XXVII., Appendix*, p. 512.[1] The chest should be examined by percussion and auscultation, and special inquiry made as to previous hæmoptysis; a single occurrence of this symptom, Sieveking thinks, should be met by an addition of fifteen years to the age, and the life should be altogether rejected if examination shows decided evidence of the existence of tubercular deposit.

Nervous system.—The principal symptoms indicating existence of actual disease of this system are paralysis, want of co-ordinating power, hyperæsthesia, anæsthesia, and certain affections of the special senses. A tendency to disease of this system, again, may be indicated by repeated attacks of giddiness or headache, or by a general appearance of plethora, accompanied by shortness of the neck. Disease of the spinal cord, a previous attack of apoplexy, or confirmed epilepsy, render the life uninsurable. Previous attacks of other diseases, *e.g.*, sunstroke, impair expectation in proportion to the amount of persisting damage. Woodman and Tidy note that sexual incapacity in males is an early symptom of many neuroses.

Circulatory system.—The pulse should be regular, between (in adults sitting) 70 to 85 per minute, soft, but not too compressible. Change of posture should not make a difference of more than 10 beats per minute. The heart sounds should be normal, and the apex-beat in the fifth intercostal space about 1½ inches below, and the same distance to the right of, the left nipple. If a murmur exists, and there is reason to suppose it to be not due to organic disease, postponement of the assurance should be advised. If due to valvular disease, its intensity affords no indication of the amount of danger. Certain forms of valvular disease damage expectation more than others. Aortic regurgitation, from liability to sudden death, is the most serious; mitral disease, especially if regurgitant in character, is much less serious. Fatty degeneration of the heart obviously greatly impairs expectation.

Digestive system.—Under this head the appearance should be noted of the tongue, lining membrane of the mouth, skin, and conjunctiva. Inquiry should be made as to the state of the appetite and action of the bowels, and as to present or previous existence of piles, jaundice, hæmatemesis, chronic vomiting, and symptoms of dyspepsia generally. Inquiry should also be made as to previous attacks of malarious disease, and an endeavour made to ascertain the condition of the spleen. Enlargement of the liver (except when due to simple congestion) renders complete rejection of the life advisable. If due to simple congestion, the examination should be postponed until the liver has recovered its normal dimensions.[2]

Genito-urinary system.—Œdema or puffiness, especially of the eyelids, hands, feet, or scrotum, should be looked for, and inquiry made as to the

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[1] Roughly, from this table the average circumference of the chest in male Europeans 5 feet 6 inches in height equals 37½ inches, and rather more than ½ an inch less for every inch below, and about ½ an inch more for every inch above this height. Approximately in the same, the vital capacity equals 190 cubic inches for a height of 5 feet 3 inches, and about 8 cubic inches more for every additional inch in height.

[2] Sieveking, *op. cit.*, p. 169.

existence of lumbar pains or dysuria. Inquiry should also be made as to the existence or otherwise, in males, of sexual incapacity and urethral stricture; and in females of symptoms indicating ovarian or uterine disease. The urine passed during twenty-four hours should be examined; its quantity should be forty to sixty ounces, specific gravity 1015 to 1025, and it should be free from blood, sugar, and albumen. Persistent presence of any of these renders the life uninsurable. The urine should also be examined for the presence of bile pigments, pus, tube casts, and crystalline deposits.

Colour blindness.—Special examination should be made for this in the case of sailors, railway guards, locomotive engine drivers, or others whose occupations are such as to expose them to danger should they mistake the colour of a signal. The form of the affection may be inability to distinguish red, or green, or violet, constituting, as the case may be, red blindness, green blindness, or violet blindness. Of these three forms the first is most and the last least common. Or the inability to distinguish these colours may be incomplete, constituting incomplete colour blindness, the most common form of all. Examination for colour blindness is best conducted by Holmgren's method. This consists in placing before the individual a number of skeins of wool of various shades of colour, and making him sort them in order according to their colour and shade.

Minor questions connected with Life Assurance are—IV. Has there been misrepresentation or concealment of material facts? V. Has the death of the assured taken place? VI. Did the assured kill himself? VII. What was the cause of the assured's death?

Question IV.—Has there been misrepresentation or concealment of material facts?

This question arises when liability to pay the sum assured is disputed on the ground that the policy is void, owing to such misrepresentation or concealment. In regard to it, it may first be stated that, in general terms, a material fact is anything the assurers have a right to be informed of, which may influence the rate of premium, irrespective of whether the party from whom the information is required, does or does not know it may have this influence, and irrespective of whether the fact is or is not a condition of things which has actually shortened the life of the assured.

The question whether a given fact is material or not may be one on which expert evidence is not required, *e.g.*, previous rejection of the life by another assurance office. Frequently, however, the fact alleged to be material is the existence at the time of effecting the assurance of a particular habit or disease, or previous attack of disease. Here the question whether the fact is material or not may depend on whether or no such habit or disease is one which usually tends to shorten life, and when this is the case the opinion of a medical expert may be required in order to enable the Court to decide the question at issue. **The existence of disease, or of a previous attack of disease, may or may not be a material fact.**—It is a material fact if information regarding its existence has been specifically required by the assurers. Information regarding the existence of the disease in question may not, however, have been specifically required, in which case the question whether the fact of its existence is material or not may turn on whether or no it is one coming within the description of a

"disease tending to shorten life."[1] On this point it has been decided[2] that when the question is one of material concealment in life assurance, only such diseases come within this description as are of a serious nature, and the usual course of which is to shorten the duration of life. **The existence of a habit such as usually tends to shorten the duration of life is obviously a material fact.** Commonly there is no difficulty in answering the general question whether or no a particular habit comes within this description. In one important case, however,[3] the expert witnesses called at the trial differed greatly in opinion on the question whether or no opium-eating was a habit tending to shorten life. The same question has arisen in regard to vegetarianism, and it is possible that it might also arise in regard to tobacco smoking. Where, however, the habit is one which, like the two last mentioned, is not usually held to shorten life, the question whether its existence is a material fact or not, has been held to depend on whether or no specific inquiry as to its existence has been made by the assurers. Considerable difficulty is sometimes met with in coming to a conclusion as to whether or no a particular habit existed at the time the assurance was effected. In the case, for example, of alleged intemperate habits, it is difficult to draw a line between moderate use and abuse of alcohol; and often difficult, therefore, to come to a conclusion as to whether what existed at the time of effecting the assurance, was the latter or the former. When, however, this is the question at issue, the decision in the case usually rests on the ordinary evidence produced, not on the expert evidence.

To establish that there has been misrepresentation or concealment, the existence at the time of effecting the assurance, of the fact alleged to have been misrepresented or concealed must be proved. This, in many cases, is a matter of ordinary, not expert, evidence. In other cases proof of the existence of the fact concealed rests in whole or in part on expert evidence; *e.g.*, the assured may have endeavoured to conceal a previous attack of disease, by concealing the name of his usual medical attendant. Such cases usually present no difficulty. Cases more difficult to deal with are (1) cases where the only evidence available of the existence of a disease tending to shorten life, is evidence of the existence of certain symptoms which may or may not have been due to the disease in question; and (2) cases where the inference, that a particular disease tending to shorten life existed at the time of effecting the assurance, rests on the rapidly fatal termination of the case from such disease. No general rules can be laid down for guidance in cases of this kind. In each case a medical witness must be guided by his knowledge of the symptoms and usual course of the disease alleged to have existed.

Question V.—Has the death of the assured taken place?

This question may in effect be one of identity, viz. Is this body that of the assured? or, in cases where no direct proof of death is obtainable, one of presumption of death. These two subjects have already been considered (see *ante*, pp. 18 and 28). Question V. also arises occasionally in cases where, with the view of defrauding an insurance company, an assured disappears, having first fabricated evidence of his own death.

[1] The questions regarding the present or previous existence of disease, put by assurance offices to an individual proposing to assure his life, after specifically mentioning various diseases, usually conclude with the words "or any other disease or disorder tending to shorten life."

[2] *Watson* v. *Mainwaring*; see Taylor, ii., p. 598.

[3] The suit regarding payment of sums assured on the Earl of Mar's life (1831). The assured was an opium-eater, but this had not been made known to the assurance company (see Christison on Poisons, p. 716).

Question VI.—Did the assured kill himself?

Assurance policies almost always contain a clause exempting the company from liability should the assured "**die by his own hands**" or "**commit suicide.**" In giving evidence at an inquest, therefore, it should be kept in mind that an opinion as to the cause of death, given at such inquest, may afterwards be called in question in an action to recover assurance money. In such a case, omission to make a thorough examination of the body, may place the witness in a very awkward position. Again, a question, which has more than once arisen in the course of actions of this kind, is this: The assured kills himself during an attack of insanity; is this "death by his own hands" or "suicide," as the case may be, according to the phrase used in the policy? On this question the English judges have expressed different opinions. It has, however, been decided by a majority of the judges, that these phrases, as used in assurance policies, include all cases of intentional self-killing, no matter whether the individual be or be not insane at the time.

Question VII.—What was the cause of the assured's death?

Obviously, when it is alleged that the assured killed himself, this question directly arises. So, also, this question may arise indirectly, in a case where it is alleged that there has been material concealment, seeing that the cause of death may afford corroborative evidence of the fact that there was such concealment. Further, the question, What was the cause of the assured's death? may arise in the following cases.

(1) In cases where the life is **assured against accident only.** Here the question takes the form: Was death due to accident or to natural causes? the assurers being liable in the former case, but not in the latter. When the cause of death has been definitely ascertained, there is usually no difficulty in coming to a conclusion on this point; and it may here be mentioned that it has been decided, that death from sunstroke does not come within the meaning of death from accident, as used in such policies. So, also, there is usually no difficulty when death occurs within a short time after the alleged accident. Where, however, a considerable interval of time has elapsed between the accident and death, difficulty may be experienced in coming to a decision on the question. In such a case, points for consideration are: (1) Were the symptoms and post-mortem appearances present in the case, such as indicate the presence of disease? (2) Could such disease have arisen from the accident? (3) What influence would the accident be likely to exert on such disease? &c., &c.

(2) **Assurance murders.**—Cases have occurred something of the following kind: A. assures B.'s life or induces B. to assign to him a policy of assurance on his (B.'s) life, and subsequently murders B.—usually by poison—afterwards claiming the sum assured. De Pommerais' case (see *Digitalis*, p. 295) was a murder of this kind. Of course, in such cases, the question, What was the cause of death? is of special importance. With the view of preventing crimes of this description, 14 Geo. III. c. 48 enacts that no assurance shall be valid, if effected on the life of another by any one who cannot show that he has a direct legitimate interest in the life of the person assured.

CHAPTER XXXIV.

INSANITY.

Causes of Insanity.

The chief causes leading to such impairment or derangement of the functions of the nerve-centres as constitutes insanity are: 1. **Original difference** in their constitution. 2. **Abnormalities in the quantity or quality of the blood** supplied to them. 3. **Excessive functional activity.** 4. **Injury or disease**: and 5. **Sympathy or reflex action.**

1. **Original difference in constitution.**—This may show itself as arrest of development, occurring before or soon after birth, giving rise to **amentia.** In such cases there may or may not be visible head deformity. The arrest of development may be consequent on changes interfering with the growth of the skull bones, as in **cretinism.** (See *Amentia.*)

2. **Abnormalities in the quantity or quality of the blood** supplied to the nerve-centres. Under this head may be classed insanity caused by long-continued bodily disease, or by the long-continued use of certain poisons, *e.g.*, alcohol or cannabis. Alcoholic intemperance appears to be the cause of insanity in about 12 per cent. of the admissions into English and American asylums; and, according to Baboo Kanny Loll Dey, in more than 38 per cent. of the admissions into the Bengal asylums, during the five years ending 1867, the insanity could be traced to the use of cannabis.[1] Insanity, the result of hereditary taint, may be classed under this or under the previous head. Hereditary predisposition is traceable, according to some writers, in 25 per cent.; according to others, in 60 per cent. of cases of insanity (see *Life Assurance*, p. 387).

3. **Excessive functional activity.**—The following are the more common causes of insanity classable under this head. Excessive trouble or grief (Bucknill and Tuke assign 14 per cent. of cases to domestic troubles or grief)[2]; religious anxiety or excitement; excessive sexual indulgence or self-abuse; and mental overwork. Less common causes are sudden joy, fear, fright, &c.

4. **Injury or disease.**—Epilepsy, apoplexy, sunstroke, external

[1] See, however, Cannabis, *ante*, p. 262. [2] Psychol. Med., p. 100.

injury to the head, may all give rise to insanity. Bucknill and Tuke calculate that epilepsy is the cause of about 6 per cent. of the admissions for insanity into asylums.[1]

5. **Sympathy or reflex action.**—Affections of the uterus and its appendages, *e.g.*, disordered menstruation, and diseases and displacements of the uterus, sometimes give rise to insanity. Pregnancy has been accompanied by insanity, the patient recovering after delivery. Insanity has also been traced to puberty, to lactation, and to the "change of life" (climacteric insanity); and has arisen from intestinal irritation.

Certain terms used to denote different varieties of insanity are derived from the supposed origin of the affection. Terms of this description in common use are puerperal insanity, epileptic insanity, toxic insanity, senile insanity, &c., &c.

Forms of Insanity.

For medico-legal purposes it is convenient to distinguish three leading forms of insanity, viz.: **I. Amentia,** or insanity due to arrest of development of the nerve-centres; **II. Dementia,** or insanity mainly characterised by an apparent failure of power of the nerve-centres; and **III. Mania,** or insanity the leading character of which is disorder of the functions of the nerve-centres. Before, however, describing these and their sub-forms, it is necessary to say a word or two on the subject of illusions and delusions, and advisable also to refer to certain phenomena of somnambulism.

Illusions and delusions.—The term "an illusion" is commonly used to denote an effect apparently solely due to a disorder of the functions of sensorial perception. In an illusion as thus defined, the limitation of the disorder to these centres, and the non-participation therein of the higher or intellectual nerve-centres, is shown by the affected individual being fully aware of the falsity of the impression arising from the disorder. Illusions vary in character according to the particular sensorial perception affected. Frequently this is visual perception, in which case we have "spectral illusions." Illusions of other senses may however occur; *e.g.*, of hearing, smell, &c. (see *Cases* D 1 and 2). The existence of an illusion as thus defined does not of itself indicate disorder of the intellect, and does not therefore of itself indicate insanity. On the other hand, the term "a delusion" is commonly used to denote an effect resulting from disorder of the functions of the higher or intellectual nerve-centres, by reason of which an individual believes in the existence of facts which have no existence in reality. Delusions are very varied in character (see *Intellectual Mania*): their existence indicates

[1] Psychological Medicine, p. 103.

insanity. Further, as delusions are due to disorder of developed intellectual power, they are not present in complete amentia, *i.e.*, insanity due to arrest of development of the nerve-centres, in its most complete form.[1]

Somnambulism.—In this condition (see *Cases* D 3 to 9) the higher or intellectual nerve-centres appear to be in a state of partial activity only; or, as in the higher form of somnambulism, in a state of full activity to one train of impressions, but inactive as regards others. In this condition very elaborate acts may be performed. Hence the mere fact of the performance of such an act, does not of itself indicate that the higher or intellectual nerve-centres were in full activity at the time of its performance. This is obviously of much medico-legal importance, seeing that such acts, done during a condition of partial activity only, of these higher centres, may result in the death or injury of others, and form the subject of a criminal inquiry.

I.—Amentia.

This may be defined as insanity due to arrest of development of the nerve-centres. In this form of insanity the individual is of unsound mind from birth. Hence amentia corresponds to what legal writers call "**dementia naturalis.**" Two forms of amentia are recognised by medical writers, viz.: 1. Complete amentia, or idiocy; and 2, Partial amentia, or imbecility.

1. **Complete amentia, or idiocy.**—In this form of amentia the arrest of development not only affects the higher or intellectual nerve-centres, but appears also to affect the centres of sensorial perception. Hence, in the fully-developed form of complete amentia, the individual carries on a mere vegetable existence, not having the sense even to eat or drink. In the more common and less developed form there is a certain amount of intelligence; the individual recognises his friends, is capable with extreme difficulty of acquiring a certain amount of education, and is able to make his wants known by signs, or imperfectly articulated words. In almost all there is visible bodily deformity, the cranium is small, its vertex depressed, and the forehead retreating. The face seems to occupy the whole of the front part of the head, the expression is vacant, there is often a squint, or deafness, and sometimes hare-lip. Their habits are often disgusting, they have no proper sense of taste or smell; they eat or drink everything, filthy or not, and pass their evacuations insensibly. There are no delusions.

2. **Partial amentia, or imbecility.**—It is difficult to draw

[1] Some writers apply the term "a hallucination" to a delusion supposed by the patient to be "produced by an external impression, although no material object acts upon his senses at the time" (Taylor).

any precise line of demarcation between partial and complete amentia. In imbecility, however, there is not that marked want of development of the centres of sensorial perception which is present in idiocy. Guy and others regard possession of the faculty of speech as the character distinguishing imbecility from idiocy. Two forms of imbecility have been described, viz : (1) **intellectual imbecility**, and (2) **moral imbecility**. Of the two, intellectual imbecility is the form which most closely approaches to idiocy in its characters, the affected individuals in well-marked cases only differing from those suffering from complete amentia in its less pronounced forms, in possessing the power of speech. Intellectual imbeciles, although markedly deficient in general intellectual power, are capable of acquiring an amount of education sufficient to fit them for carrying on duties requiring no great mental effort (see *Cases* D 10 and 11). In moral imbecility the defective development appears to affect chiefly the higher functions of the brain, the affected individual, although fairly intelligent and shrewd, being seemingly deficient in moral sense and in power of self-control (see *Cases* D 12 and 13), his mental condition in some cases closely approaching to that present in moral mania. It may further be noted that in some cases of imbecility the individuals are greatly "under the dominion of childish fancies,"[1] approaching in character to delusions (see *Case* D 14).

Cretinism.—This is the name given to a form of amentia prevalent in certain districts, and apparently the result of local conditions. It is largely met with in Switzerland and Savoy, and very probably depends on the occurrence of changes interfering with the growth of the skull bones. It is usually associated with enlargement of the thyroid gland, or goitre.

II.—Dementia.

Legal writers use this term as synonymous with insanity, grouping all cases of mental alienation under the two heads of (1) **Dementia naturalis**, *i.e.*, individuals insane from birth; and (2) **Dementia adventitia**, or **accidentalis**, *i.e.*, individuals who become insane after birth. In **medical phraseology**, the term **dementia** is employed to denote that form of insanity in which the mental powers, having attained maturity, subsequently fail; the individual falling into a condition more or less resembling amentia, but distinguished from amentia, by being the result of failure of power previously present, and not the result of original want of power. Dementia may be **acute**, *i.e.*, come on suddenly; or **chronic**, *i.e.*, come on slowly; and may be **secondary**, *i.e.*, follow on a previous attack of mental or other disease; or

[1] Guy, For. Med., p. 184.

primary, *i.e.*, come on unpreceded by any such attack. In comparatively rare cases, dementia is both acute and primary.[1] In others it is acute, but secondary to a serious attack of brain or other disease; *e.g.*, epilepsy, apoplexy, typhus or typhoid fever, acute rheumatism, &c. Recovery frequently takes place from acute dementia. Usually dementia is chronic, and may then either be primary, coming on as a result of drunkenness, masturbation, or excessive sexual indulgence; or be secondary to a previous attack of mania; or supervene as the result of old age (senile dementia). When dementia comes on slowly, often the first symptom noticed is failure of memory. This is followed by general dulness of all the mental faculties. The bodily health is usually good. In very advanced cases the functions of the centres of sensorial perception become impaired—indeed, insensibility to pain is often noticed in the early stages—and the animal instincts even are lost. Recovery rarely, if ever, takes place from chronic dementia. Dementia may be accompanied by occasional attacks of maniacal excitement.

General paralysis of the insane.—This is the name given to a form of dementia, in which the failure of the power of the higher or intellectual nerve-centres is accompanied by failure of power of the motor centres. General paralysis is rare in women. It most commonly attacks men of education and position. In the majority of cases of general paralysis, the failure of memory and of the intellectual powers generally—usual in dementia—is accompanied by delusions of possession of exalted power and boundless wealth. Along with these symptoms indicative of affection of the higher nerve-centres, impairment of power—first noticeable in the tongue and muscles of articulation—is observed, indicating affection of the motor centres. The pupils become irregular; the power of precise co-ordination of movement necessary for the performance of what may be called acquired automatic acts, *e.g.*, walking, is lost; and general impairment of motor power supervenes. Apparently the centres of sensorial perception, as a rule, do not become markedly affected until near the end of the case, but, as in chronic dementia, deficient sensibility to pain may be an early symptom (see *Case* D 16). This deficient sensibility to pain is sometimes of medico-legal importance (see *General Intellectual Mania*).

III.—Mania.

Under this head may be classed all forms of insanity characterised by disturbance or disorder (as distinguished from want of development, or failure) of the functions of the higher nerve-

[1] In India acute primary dementia, always rare, is, when met with, generally a result of sunstroke.

centres.[1] Unlike amentia and dementia, **mania is seldom continuous**, there being usually remissions, more or less complete. If complete, a remission constitutes what is termed a **lucid interval** (see *post*, p. 406). Mania may come on suddenly or slowly; if slowly, certain **premonitory symptoms** are usually first noticed. The chief of these are indigestion, constipation, and sleeplessness; altered or perverted sensations, sometimes amounting to illusions; great irritability, alterations of temper, disposition, and habits, and inability to concentrate the attention on any train of thought. According to Dr. Radcliffe, the leading mental characteristics in incipient insanity are:—1. Self-conceit, the individual fancying himself wiser, richer, or stronger than he really is. 2. Misanthropy, or general dislike to others without cause; and 3. Suspicion, often leading to delusions of the existence of conspiracies to injure or poison the sufferer.[2]

Using the term "mania" in the extended sense stated above, various forms of it are recognised; these are usually classed according as to whether—(*a*) **Delusions** are present.* If present, the affection is classed as intellectual or ideational[3] mania; if absent, as moral or affective mania. (*b*) The mental functions affected are **disordered generally, or in one particular** only. If the former, the affection is classed as general; if the latter, as partial. This method of classification (Ray's) gives, therefore, four chief forms of mania, viz., 1. General intellectual; 2. Partial intellectual; 3. General moral; and 4. Partial moral. A further subdivision of some, at least, of these four forms is possible, according as to whether (*c*) **Depression or excitement** is present. If the former, the affection is classed as melancholic; if the latter, as non-melancholic.

It should, however, be remarked, that although many cases of mania are so distinct in type as to be readily classable under one or other of the above-mentioned forms, in many such distinction of type is absent.

1. General Intellectual or Ideational Mania.

In this form of mania there appears to be general disorder of the functions of the higher nerve-centres. It is divisible into a non-melancholic form and a melancholic form, according as to

[1] Some writers on Insanity limit the application of the term "mania" to one particular form of mental disorder, viz., to that in which there is general disorder of all the intellectual powers coupled with excitement—what, in fact, when the term "mania" is used in the wider sense above stated, may be called the non-melancholic form of general intellectual mania.

[2] Taylor, ii., p. 467.

[3] The term "ideation" has been used to denote a function of the higher nerve-centres, of the nature of sensorial perception, but higher in character. Disorder of ideation, using the term in this sense, results in delusions: hence the term "ideational mania."

whether excitement or depression is present. Some writers on Insanity limit the application of the term "mania" to the non-melancholic variety of this form of insanity, and apply the term general melancholia, or lypemania, to the melancholic variety. Sometimes the two forms blend, excitement and depression alternating with one another in the same case.

The principal symptoms of general intellectual mania are: Rapid flow of ideas, expressed with confusion and incoherence; the attention is constantly wandering, and delusions rapidly succeed one another. In one form the individual fears everybody and everything (panophobia); in another he imagines himself pursued by demons (demonomania). There may be furious excitement; or, in the melancholic form, great apparent suffering and depression. The muscular power is often much increased, and the patient is often violent and very destructive, rendering great caution necessary in visiting him. The expression is altered, he sleeps but little, and there is often (especially if the case is tending towards general paralysis) deficient sensibility to pain (see *Cases* D 15 and 16). The fact that in this form of insanity there is often diminished sensibility to pain, may be of importance in cases where injuries received by insane persons form the subject of an inquiry: *a'*, from its indirectly tending to increase the amount of injury likely to be inflicted during a struggle; and, *b'*, as bearing (as in *Case* D 16) on the question of the time of infliction of an injury.

2. Partial Intellectual or Ideational Mania, or Monomania.

Some writers confine the use of the term "monomania" to the non-melancholic form of this variety of insanity, and call the melancholic form partial melancholia or lypemania. The leading character of this form of insanity, may be stated to be the affection of ideation as regards one particular only. Hence there is either only one delusion, or, in more developed cases, a series of delusions, connected together by one morbid idea (see *Case* D 17). The delusion may be of the most ridiculous character; the individual may believe himself to be made of glass, or to be dead, or to be some celebrated character. In the melancholic form of monomania the delusion, or delusions, are frequently of a religious character (religious monomania), or, as in *Case* D 17, delusions of persecution (monomania of persecution). Such delusions may lead to the commission of homicide (see *Cases* D 18 and 19), or to suicide.[1] In markedly distinct cases of monomania, the individual appears to be perfectly sane on all points

[1] Monomaniacs, in fact, may, under the influence of their delusions, exhibit propensities similar to those exhibited, without delusion, in the various forms of partial moral mania (see *post*, p. 403).

unconnected with his delusion or delusions, and only betrays excitement or depression when these are touched upon. In such cases (especially in non-melancholic cases), the individual may appear to reason correctly and accurately on matters unconnected with his delusions, and even in matters connected with them his reasoning may be accurate, although his conclusions, being founded on false premises, are erroneous. Sometimes in these cases, particularly if the individual has any powerful motive for concealing his delusion, there may be great difficulty in detecting its existence (see *Cases* D 20 and 21). In other cases, especially advanced cases, the reasoning powers appear generally affected, so that it becomes difficult to decide whether the case is one of partial, or one of general ideational insanity. Mónomaniacs are often readily imposed upon and controlled by a person affecting to believe in their delusions (see *Cases* D 21 and 22).

3. General Moral or Affective Mania.

Moral or affective mania (mania sine delirio) is distinguished from intellectual or ideational mania by the absence of delusions. It may best be defined, in the words of Ogston,[1] as "consisting in a morbid perversion of the natural feelings, affections, inclinations, temper, habits, moral dispositions, and natural impulses, without any remarkable disorder of the intellect, or knowing and reasoning faculties." In general moral mania, this morbid perversion is general, and, as a consequence, the individual exhibits several morbid propensities. *Cases* D 23 and 24 are typical examples of this form of mania.

4. Partial Moral or Affective Mania.

This form of mania only differs from the preceding variety in the fact that the morbid perversion is not general, but limited to one or two particulars. Hence, in partial moral mania, the individual exhibits one or two, instead of several, morbid propensities. Under this form of mania may be classed the impulsive or instinctive insanity of some writers. Different varieties of partial moral mania are distinguished, according to the morbid propensity present, *e.g.*, homicidal mania, suicidal mania, kleptomania, pyromania, &c. Medico-legally the more important are the following:—

1. **Homicidal mania.**—It has been already pointed out that, as in cases D 18 and 19, homicide may be the result of the existence of a delusion. Such cases, adopting Ray's classification of mania, belong to intellectual insanity, usually to the partial form, and may therefore be called cases of homicidal monomania.

[1] Lectures, p. 304.

In some cases, however, the homicide, or attempt at homicide, appears to be the result of an insane propensity or impulse, unaccompanied, at least so far as can be ascertained, by a delusion. These, adopting Ray's classification, must be classed as cases of moral or affective mania, usually of the partial variety, and to these the term homicidal mania is commonly applied. In some cases the insane propensity appears to be of gradual growth (see *Case* D 25); in others, previous to the commission of the act, the individual shows symptoms (perhaps only slight symptoms) of the existence of mental disorder (see *Case* D 26), and it has been noticed that homicidal tendencies may co-exist with a quiet exterior (see *Case* D 27). In other cases, again, the homicidal act appears to be the result of a sudden and uncontrollable impulse, occurring in an apparently sane person, the commission of the act being, as it were, the only symptom of insanity exhibited (see *Case* D 28). Not infrequently the homicidal propensity or impulse appears to be connected with disordered menstruation, as in *Case* D 26; or with parturition, or with epilepsy. Not infrequently, also, it is accompanied by suicidal tendencies. Especially in cases where the symptoms of insanity present are slight, importance attaches to the character of the act. The chief points usually stated to be indicative of homicide by an insane are:—

(*a*) **The absence of motive.**—*Case* D 26 is an example of this. Sometimes there is not only an entire absence of motive, but, as pointed out by Taylor, the act is done "in opposition to all human motives." A woman, for example, murders her children (see *Case* D 18), or a man, known to be fondly attached to his wife, kills her. Caution, however, is necessary in judging from this character. In a murder by a sane person there may be an apparent absence of motive, simply because the motive has not been discovered. On the other hand, in cases of homicide by undoubtedly insane persons, a motive—often, it is true, incommensurate with the act—has existed, or has appeared to exist. Again, in cases of homicide by sane persons, especially in India, the motive leading to the crime is sometimes a very trivial one.

(*b*) **The absence of any attempt at concealment** of the act. —*Cases* D 26 and 28 afford examples of this. On the other hand, *Case* D 12 is an example of considerable effort at concealment of homicide by an insane.

(*c*) **The absence of accomplices.**—This character is often present in homicide by sane persons. The existence, however, of accomplices strongly indicates sanity.

(*d*) **Numerous murders committed at the same time.**—Little reliance can, however, be placed on this character. In homicide by insanes there is often only a single victim (see *Cases* D 12, 19, and 26). On the other hand, in homicide by sane persons, there are sometimes numerous victims (see *Running amok, ante,* p 263).

2. Suicidal mania.—Suicide by an insane may, like homicide, result from a delusion (suicidal monomania), or may result simply from the existence of an insane propensity or uncontrollable impulse for self-destruction, unaccompanied by any discoverable delusion (suicidal mania). A tendency to suicide often co-exists with homicidal tendencies. Suicide without any discoverable motive does not necessarily amount to proof of the existence of insanity, although it to a certain extent indicates it.[1]

3. Kleptomania.—In some cases theft committed by an insane is distinctly traceable to the existence of a delusion, *e.g.*, the individual may believe that he is only recovering property stolen from him. This sometimes occurs as an outcome of the delusions of boundless wealth often present in incipient general paralysis. Or, again, the individual may believe that he has received a Divine command to take possession of the articles he steals. In other cases, by no means common, there is no delusion; but simply a morbid propensity, or uncontrollable impulse to steal or to acquire. Kleptomania is sometimes strikingly hereditary,[2] and it is alleged that it has often shown itself in women labouring under disordered menstruation, or far advanced in pregnancy.[3] Browne ('Medical Jurisprudence of Insanity,' p. 132) goes at length into the characters which distinguish theft by kleptomaniacs, from theft by sane persons. A brief summary of these is as follows:—1. The articles stolen are such as the means of the individual would readily enable him to purchase (see *Case* D 29), or are of little value. 2. Some kleptomaniacs steal openly, others willingly avow the act, or restore the goods stolen. Some, however, conceal the theft with much ingenuity. 3. Kleptomaniacs, as a rule, make no use of the articles stolen; they either throw them away or hoard them, and have no accomplices. 4. In many instances, but not invariably, the articles stolen are bright and glittering articles. *Case* D 30, in which kleptomania was set up as a defence to a charge of theft, illustrates the points to be attended to in forming an opinion on cases in which it is alleged this form of insanity exists.

4. Pyromania.—Cases of pyromania, or morbid propensity for incendiarism, sometimes occur. Young females, suffering

[1] The verdict "suicide while in a state of temporary insanity," so frequently returned by coroners' juries in England, is most probably in many cases the result of the fact, that by the law of England, self-destruction (in a person of sound mind) is a felony (*felo-de-se*), entailing forfeiture of goods and burial in unconsecrated ground. The law of India, however, contains no provision making the actual commission of suicide an offence, although an attempt to commit it is (see Wounds, p. 55). Sect. 30 of the Coroners Act (IV. of 1871) expressly declares, that it shall no longer be the duty of coroners in India to inquire whether any person dying by his own hand was or was not *felo-de-se*, and further that a *felo-de-se* shall not forfeit his goods.

[2] Bucknill and Tuke, p. 284.

[3] Marc and others, quoted by Taylor, Manual, p. 757.

from disordered menstruation, hysteria, or epilepsy, are said to be specially liable to it.

5. **Other forms of partial moral mania** mentioned by writers on insanity are **Erotomania**, or a morbid propensity to amorous ideas. **Dipsomania**, or a morbid propensity to indulge in intoxicants. **Nostalgia**, or home sickness, &c., &c. As regards the first, it may be mentioned that when the morbid propensity originates in the organs of reproduction it is called **Nymphomania** in females, and **Satyriasis** in males. The second, Dipsomania (also called **Oinomania**), might possibly be pleaded as a defence to a criminal charge; it would, however, in such a case, have to be established that the propensity was **irresistible**, always an extremely difficult matter. The third, **Nostalgia**, is usually accompanied by depression, and, like **Hypochondriasis**, may be classed as a melancholic form of this variety of mania.

In partial amentia or imbecility, morbid propensities similar to those present in partial moral mania may exist (see *Case* D 31, and also *Case* D 12).

LUCID INTERVALS.

By "a lucid interval" is meant a complete intermission, not, merely a remission, of the mental disorder. Some authorities hold, that lucid intervals, as thus defined, do not exist, because, (*a*) if the intermission is absolutely complete, the patient is cured; while (*b*) if a predisposition to the return of the disease exists, the patient cannot be said to be free from the disease, and his condition must be regarded as one of remission—such as are common in mental diseases—but more complete than usual. Whether, however, lucid intervals are complete intermissions or not, it is undoubtedly the fact that, in some forms of mental disorder, intervals of apparent sanity occur. These are what are known as lucid intervals. Lucid intervals never occur in amentia. They sometimes occur in dementia, when this is secondary to a previous attack of mania. They do not occur in chronic dementia, but in this form of dementia, a temporary brightening of the faculties sometimes takes place just before death. **Lucid intervals are common in all forms of mania**, they may supervene suddenly, or come on gradually, and may last for a few minutes, or may be prolonged for years. In a perfect lucid interval, an individual should be able to talk rationally about his symptoms, and describe his delusions as delusions, betraying no excitement. It is sometimes, however, extremely difficult to discover whether or no a lucid interval proper is actually present. What appears to be a lucid interval, may, in fact, be simply a remission, accompanied by greater exercise of self-control than usual.

EXAMINATION OF ALLEGED INSANES.

Indications of the existence or otherwise of insanity may be derived from—1. The general appearance of the patient; 2. His bodily condition; 3. His history; and 4. His mental condition and capacity.

1. **The general appearance of the patient.**—Under this head should be specially noticed: (*a*) any cranial deformity (see Amentia); (*b*) the facial expression and gestures,—these are often highly indicative of insanity, especially of its advanced or more fully-developed forms; and (*c*) any peculiarities of dress, gait, or surroundings.

2. **His bodily condition.**—Note specially: (*a*) The condition of the digestive functions; these are often disordered in the early stages of insanity, the skin becoming harsh and dry: (*b*) The state of the pulse, and the presence or absence of febrile symptoms; this is important in distinguishing between insanity and the delirium of disease: and (*c*) The presence or absence of insomnia, restlessness, excitement, depression, or defect of speech or articulation. Bucknill and Tuke observe that in a great many cases of chronic mania, the hair becomes rough and bristling. A blood tumour of the ear ending in shrivelling, the so-called asylum or insane ear, is often noticed in advanced cases.

3. **His history.**—(1) As indicative of the **cause**; (2) as indicative of the **existence** of the disease. As to (1), a reference to what has been stated regarding the causes of insanity, will show the importance of inquiring into the existence or absence of (*a*) congenital defect, (*b*) hereditary taint, (*c*) habitual indulgence in intoxicants, (*d*) disorders, especially in females, of the reproductive organs, (*e*) epilepsy, or other brain affection or injury, (*f*) excessive sexual indulgence, and (*g*) mental overwork, anxiety, or sudden shock. Inquiry should also be made as to whether anything has occurred likely to induce the individual to feign insanity. It must not be forgotten, however, that as in *Case* D 32, insanity may arise from the anxiety of mind resulting from a criminal charge. As to (2), it should be noted whether or no (*a'*) there has been any previous attack of insanity; (*b'*) there has been any marked alteration or change in the feelings, affections, and habits of the patient; and (*c'*) inquiry should be made generally as to the symptoms observed at the commencement of the alleged outbreak of insanity.

4. **His mental condition and capacity.**—Inference as to this may be drawn from the patient's (1) answers to questions, (2) acts, and (3) writings. As regards (1), the patient's memory may first be tested. He may be asked, for example, his name, place of birth, as to the occupation of his parents, number of brothers and sisters or children, the date, the names of well-known

persons, and may be asked to count in order from one upwards, &c. Next, his judgment may be tested; he may be asked to perform simple arithmetical operations, may be questioned as to his knowledge of the value of money, and generally as to the inferences he would draw from particular facts. While questioning him, his power of fixing his attention should be observed. Next the existence of delusions should be searched for: if these are known, the conversation should be led to them; if not, the conversation should be led to various topics in succession (see *Case* D 20). Lastly, the state of the moral feelings should be inquired into by directing the conversation to the subject of the patient's friends and relatives. This testing of the mental capacity by questions, is of special importance in cases of supposed feigned insanity. Except in complete amentia, advanced dementia, or possibly also in an actual paroxysm of maniacal excitement; in true insanes, consciousness, memory, and reasoning power, especially as regards matters unconnected with their delusions, remain, at any rate, to a certain extent, intact. *Case* D 33 is an example of feigned insanity, detected by persistently silly and erroneous answers to simple questions. Care should be taken that the questions asked are not too complex, but are such as the individual under examination might reasonably, from his education and position, be expected to be able to answer. Ogston relates a case,[1] for example, in which a medical witness put forward as evidence of mental incapacity, the fact, that an alleged imbecile could not tell how much per cent. £20 interest on £1200 amounted to, though he himself (the witness), when asked to answer the same question, was unable to do so. During the course of the examination, it should be noted whether the individual, as is usually the case with impostors, appears to be trying to make himself out to be mad. True insanes will often argue with considerable ability that they are not mad. Others are conscious of their condition. A constant putting forward, however, of evidence of insanity, should always be looked on with suspicion. (2) As to the evidence of mental disorder afforded by the acts of the patient, it should be recollected that these in a true insane are the results of his disordered mental condition. Where delusions exist, his acts and antics are connected with them, even although the connection may be apparently inexplicable (see *Case* D 34). Sometimes, as Dr. Guy remarks, "the acts of the maniac evince the same forethought and preparation as those of the sane" (see *Case* D 35); and lastly, true insanes are generally easily imposed upon (see *Case* D 22). As to (3), frequently evidence of the existence of mental disorder is obtainable from examination of matters written by the patient. These may exhibit incoherence, or betray the existence of delusions; but except in cases of approaching general paralysis, the

[1] Case of David Yoolow, Lectures, p. 297.

legibility of the handwriting is not usually affected. Sometimes the approach of insanity is indicated, by a person beginning to omit words from, or spell badly in his writings.

Distinction of feigned from true Insanity.

The chief points by which feigned insanity may be distinguished are—

1. **Absence of characteristic facial expression.**—In insanity, especially in the fully developed forms usually feigned by impostors, the facial expression is characteristic. In feigned insanity, this characteristic facial expression is usually absent, or if present, is not persistent.

2. **Absence of bodily disorder.**—Bodily disorder is usually present in true, and absent in feigned insanity. The presence or absence of insomnia should specially be noted. True insanes sleep but little; impostors, exhausted by their exertions in feigning insanity, sleep soundly. Deafness and dumbness are sometimes feigned. These in true insanes are usually congenital; in feigned insanes they come on suddenly, and after the occurrence of an event, likely to induce the individual to feign insanity.

3. **Sudden attack without sufficient cause.**—In true insanity, if the attack is sudden, inquiry will, as a rule, show a sufficient cause for the attack. Feigned insanity usually appears suddenly, without sufficient cause, and is generally traceable to a desire to escape punishment (see, however, *Case* D 32).

4. **Want of uniformity in the symptoms.**—In feigned insanity, the symptoms are, as a rule, not uniform with any distinct type of the true disease. The impostor, for example, mixes general mania with excitement, with advanced dementia, &c. That variation from distinct type is often present in a case of true insanity, should, however, be borne in mind.

5. **Persistent obtrusion of the symptoms.**—Impostors nearly always try to convince you that they are mad, putting forward evidence of their insanity, especially when they think they are under observation. The fact of being under observation, makes little difference in the behaviour of a true insane.

In many cases, a satisfactory diagnosis between feigned and true insanity can only be arrived at by subjecting the patient to **prolonged observation.** It must not be forgotten also, that an expert witness, when called upon to give an opinion as to the mental capacity of an individual alleged to be insane, must be prepared, as in other cases, to state the grounds upon which his opinion is based.

CHAPTER XXXV.

LEGAL RELATIONS OF INSANITY.

It may first be remarked that in the present state of our knowledge, it does not appear to be possible to frame a thoroughly satisfactory definition of the term "insanity." One of the chief difficulties in the way of doing so lies in the fact that it is impossible to set up a standard of sanity. Any definition, for example, to the effect that insanity is mental imperfection, incapacity, or disorder, arising from certain causes, involves the setting up of such a standard. Such definitions, in fact, involve the necessity of our laying down a standard of mental perfection, capacity, or order (in other words, of sanity), deviation from which shall be held to constitute insanity. Nor is the difficulty diminished by the substitution for the term insanity, of other terms, such as unsoundness of mind, mental aberration, or mental alienation. This difficulty of definition is, however, of comparatively little importance, for the following reason. Whenever a legal right, liability, or disability, arises out of the fact that an individual is insane, it does **not arise simply out of the fact of** the individual's **insanity, but arises out of the fact** that the individual, by reason of his insanity, is—or was, at a certain specified time—**mentally incapacitated to a certain extent** or degree. The degree of mental incapacity which must be proved to exist, in order to establish that such right, liability, or disability accrues, varies with the nature of the right, liability, or disability, in question.

Hence when, in the course of an inquiry for medico-legal purposes, an individual's sanity or insanity comes into question, what has to be determined is not simply, is the individual insane, or was he insane, at a certain specified time. Were it so, a definition of insanity would be necessary. What has really to be determined is this: is this individual, or was this individual at a certain specified time, by reason of insanity, mentally incapacitated to a certain extent or degree? Such questions may arise in criminal cases, and also in civil cases. Again, also, the question frequently arises, whether or no the insanity of the individual is of such a nature as to justify his being placed in an asylum, or under restraint.

A.—CRIMINAL CASES.

I.—Criminal Responsibility.

By law every one is presumed to be of mental capacity sufficient to render him responsible for his acts. In criminal cases this presumption may be rebutted, by proof that at the time the act was done, the individual, by reason of unsoundness of mind, was mentally incapacitated to a certain defined extent or degree. The burden of proving this rests with those who assert it. We have now to consider what is this **degree of mental incapacity**, which must be proved before an individual will be held irresponsible, or entitled to an acquittal on the ground of insanity.

The English law on this subject is to be found in the answers given in 1843 by the English judges, to certain questions propounded to them by the House of Lords. These questions were put to the judges in consequence of the McNaughten case (*Case* D 36). The object of these questions was to obtain an authoritative statement of the law for the future guidance of the Courts, and the answers of the judges thereto have ever since been held to embody the law of England on the subject.

These answers are also embodied in sect. 84 of the Indian Penal Code, which constitutes the law of India on the subject of the criminal responsibility of insanes. This section is as follows: "Nothing is an offence which is done by a person who, at the time of doing it, by reason of unsoundness of mind, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law." The effect of this section may be stated to be as follows: Suppose it to be proved that an individual has done an act which, were he sane, would be an offence—say, for example, A. has killed B. Suppose, also, it to be proved that A. at the time of killing B. was insane. A. would be entitled to an acquittal if he, at the time of killing B., was by reason of insanity mentally incapacitated to one or other of the following degrees:—

1. To such a degree as to render him "incapable of knowing the nature of the act"; as, for example, if A. in killing B. did so under the insane delusion that he was slaying a wild beast or breaking a jar; or

2. To such a degree as to render him incapable of knowing that he was "doing what is either wrong or contrary to law"; as, for example, if A. at the time of killing B. was under the insane delusion that B. was attacking him (A.), for the purpose of killing him; for in that case A.'s insanity would render him incapable of knowing that he was acting contrary to law, seeing that A., were his delusion true, would be justified by law in killing B.

On the other hand, A. would not be entitled to an acquittal if

all that was proved in regard to his insanity was that he killed B. under the insane delusion that B. had blasted his character; for in that case A., even were his delusion true, would not be justified by law in killing B.; and would be presumed, the contrary not being shown, to know the nature of his act, and also that he was acting contrary to law.

Another point requiring consideration is as follows:—There is a general consensus of opinion among writers on Insanity, 1st, that one effect of insanity may be a weakening of the affected individual's power of self-control; 2nd, that in some cases the power of self-control is totally lost, the result being the production of an uncontrollable impulse—*i.e.*, an impulse which nothing short of mechanical restraint will control (see *Case* D 25)—to do certain acts; and 3rd, that such weakening or total loss of the power of self-control may occur, both in insanity accompanied by delusions, and in insanity unaccompanied thereby. The question therefore arises:—Suppose A. to have killed B., and the only thing proved about A.'s insanity is that, by reason of insanity, A.'s power of self-control was, at the time he killed B., weakened or entirely lost, what would be the legal effect?

To this question it may be answered:—

1. That any weakening short of total loss of power of self-control, would not entitle A. to an acquittal, either under Indian or English law.

2. That, according to Indian law, total loss of power of self-control would not entitle A. to an acquittal, except the Court consider it proved that, by reason of such total loss, A. at the time of doing the act was, in the words of the section, "incapable of knowing the nature of the act, or that he" was "doing what is either wrong or contrary to law."

3. As regards the law of England on this last point, Sir J. F. Stephen[1] states that it is doubtful whether or no an act is a crime if done under the following circumstances: by a person suffering from mental disease, who at the time of doing the act was by such disease totally prevented from controlling his own conduct.

Hence, in a case where the question of criminal responsibility is concerned, a medical witness should not simply direct his examination towards ascertaining whether the accused is insane or not. He should in addition endeavour to form an opinion as to whether, by reason of insanity, the accused is mentally incapacitated to the degree specified in sect. 84 of the Penal Code. He must, however, recollect that the real question at issue is the mental state of the individual at the time he committed the act. Hence he must be prepared if called upon to give his opinion as to this, and, as in other cases, must also be prepared to state the grounds on which his opinion is based. It may happen that, in order to arrive at a correct opinion, he has to take into con-

[1] 'Digest of the Criminal Law,' p. 21.

sideration not only (1) facts which he has himself observed, but also (2) circumstances which he has heard deposed to in evidence, or of which he has been informed. It is obvious, however, that any opinion based upon circumstances not within the knowledge of the witness is worthless, unless such circumstances are admitted or proved to be true in fact; and such opinion, therefore, should be given on the hypothesis that these circumstances really exist, and should be stated to depend on such hypothesis.

II.—Validity of Consent.

In certain cases the fact that an individual has given a valid consent to suffer what has been done to him, affects the question of the criminality of the doer. But by sect. 90 of the Indian Penal Code (see *ante*, p. 320), a consent is invalid if given by a person who "from unsoundness of mind, or intoxication, is unable to understand the nature and consequence of that to which he gives his consent." Hence, in certain cases, the question may arise whether a consent proved, or admitted to have been given, was or was not invalidated by the fact that at the time of giving it, the giver was mentally incapacitated to the degree specified in this section.

This question may arise in rape cases, for the consent of a female to sexual intercourse may be invalid by reason of her insanity. By the law of India, proof of insanity to the degree above stated invalidates the consent. This is not so in England, where a female, even if she be insane to the degree specified in sect. 90 of the Indian Penal Code, may yet be capable of giving a consent to sexual intercourse, sufficient to exculpate an accused from a charge of rape, and reduce the offence committed to a misdemeanour (see *Rape*, p. 321).

The same question may arise in cases where death or hurt has been caused. By the law of India, if a person over the age of eighteen suffers death or harm from an act done to him with his valid consent, the fact that he so consented may have the effect of reducing the offence committed, from murder to culpable homicide not amounting to murder;[1] or may even, if the act be one coming under the description of sect. 87 of the Code,[2] absolve the doer of the act from all criminality.

[1] Sect. 300, Exception 5, of the Ind. Pen. Code is as follows:—"Culpable homicide is not murder when the person whose death is caused, being above the age of eighteen years, suffers death or takes the risk of death with his own consent."

[2] Sect. 87: "Nothing which is not intended to cause death or grievous hurt, and which is not known by the doer to be likely to cause death or grievous hurt, is an offence by reason of any harm which it may cause, or be intended by the doer to cause, to any person above eighteen years of age, who has given consent, whether express or implied, to suffer that harm; or by reason of any harm which it may be known by the doer to be likely to cause to any such person, who has consented to take the risk of that harm."

It should also be pointed out that, by sect. 305 of the Indian Penal Code, abetment of suicide of "any person under eighteen years of age, any insane person, any delirious person, any idiot, or any person in a state of intoxication," is punishable with death ot transportation for life, while the maximum punishment awardable for abetment of suicide of a person not coming under the above description is by sect. 306 ten years' imprisonment. The degree to which a person must be mentally incapacitated, to be an insane person within the meaning of sect. 305, is not defined.

III.—Capacity of an accused to make his defence.

Another question connected with insanity which may arise in criminal cases is this: Is, or is not, the accused "of unsound mind, and consequently incapable of making his defence?" (See sects. 464 and 465 Criminal Procedure Code, *ante*, p. 5.) Obviously in such cases an expert called upon to examine the accused should direct his examination, not simply to the question whether the individual is or is not insane, but to the question whether or no the individual is mentally incapacitated to the extent indicated in these sections.

B.—CIVIL CASES.

I.—Competency as a witness.

The law of India on this subject is embodied in section 118 of the Indian Evidence Act (see *ante*, p. 10). The "explanation" attached to this section is as follows: "A lunatic is not incompetent to testify unless he is prevented by his lunacy from understanding the questions put to him and giving rational answers to them." The "competency" of a witness to testify is a matter quite distinct from the "credibility" of his evidence. Hence it may be that a lunatic who has been declared by the Court competent to testify, may give evidence which the other circumstances of the case may show, ought not to be believed. As in the case of testamentary capacity, no amount of disease of the nervous system not affecting the mind renders an individual incompetent as a witness. Thus by section 119 of the same Act: "A witness who is unable to speak may give his evidence in any other manner in which he can make it intelligible, as by writing or by signs; but such writing must be written and the signs made in open court. Evidence so given shall be deemed to be oral evidence."

II.—Testamentary Capacity.

By testamentary capacity, is meant capacity to make a valid will. To invalidate a will on the ground of the insanity of the testator, it must be proved that at the time the will was made, the

testator was mentally incapacitated to a certain extent or degree. This degree may be defined to be that he either (1) did not know the nature of the act he was performing, or (2) was not fully aware of its consequences; or (3) has made a disposal of his property which he would not have made had his mind been sound, under the influence of a delusion, or of a disorder of the mind, perverting his affections, or sense of right[1] (see *Case* D 37, *Banks* v. *Goodfellow*, and D 38, *Smee* v. *Smee*).

A person who is insane therefore may make a valid will, provided at the time of making it, he was not mentally incapacitated to the degree specified above (see *Case* D 37). A valid will may, of course, be made by a lunatic in a lucid interval. Obviously, however, the shorter the alleged lucid interval, the greater the caution which should be exercised in accepting evidence of its having occurred.

Mere eccentricity will not invalidate a will, nor will any disease of the nervous system not affecting the mind. For example, a person speechless and paralysed from apoplexy may (his mind being unaffected) make a valid will.

A medical man in examining into the testamentary capacity of an individual, might ask him to repeat the principal provisions of his will, and explain their action. Ability to do so would show that the testator understood the nature, and was aware of the consequence, of the act he was performing. The existence of delusions, &c., likely to affect the provisions of the will, should, of course, also be inquired into.

III.—Capacity of a person of unsound mind to manage his affairs.

When a person is alleged to be of unsound mind and incapable of managing his affairs, an inquiry into the truth of this allegation may, on proper application, be ordered by a Court so empowered. On such incapacity being proved, the individual may be deprived of the control and management of his property,[2] and a person appointed to manage it for him. The Courts are also empowered to make suitable provision for the protection[3] of the individual, *e.g.*, by appointing a person to take care of him. Formerly in **England** the usual procedure in such cases was to order—under what is technically called a writ "de lunatico inquirendo"—an inquiry to take place before a "commission in lunacy"; lately, however, the proceedings have been much simplified. **In India** the conduct of proceedings of this nature is provided

[1] Undue influence exerted on a person of feeble intellect may be held to render a will invalid, although the feebleness of intellect considered *per se* be insufficient to invalidate it.

[2] Placed under "interdiction" is the technical expression.

[3] Protection is distinct from restraint (see Imposition of Restraint).

for by Acts 34 & 35 of 1858. Of these Acts, Act 34 applies to the High Courts, and Act 35 to Courts other than the High Courts. By section 10 of Act 35, "the legal heir of the lunatic" is not in any case to "be appointed guardian of his person."

In all such cases the question at issue is not simply, whether or no the individual is insane; but whether or no he is mentally incapacitated to such a degree as to render him incapable of managing his affairs. That this is so must always be borne in mind while examining alleged insanes in these cases. **No general rule can be laid down** as to what should, in these cases, be held to **constitute incapacity.** Where doubt exists, it should be given in favour of sanity, *i.e.*, in favour of the supposition, from which, if established, disability does not arise. It may however be pointed out:

1. That in cases of complete amentia, advanced dementia, and general intellectual mania, the individual is obviously incapacitated.

2. That in partial intellectual mania, an individual may be incapacitated or not, according as to whether his delusion does or does not interfere with his capacity. For example, an individual may believe himself to be made of glass, and yet be perfectly competent to manage his affairs.

3. That in moral mania, especially partial moral mania, the mental disorder may well be of such a nature as not to interfere with the individual's capacity.

The cases which present most difficulty, are usually cases of incomplete amentia, especially the less developed form (moral imbecility). In such cases very great conflict of opinion often exists among the expert witnesses as to the individual mental condition and capacity. This was so in case D 13 (the Windham case). Lastly it may be remarked that defective memory arising from old age does not, *per se*, constitute incapacity. "A defective memory in an aged person taken alone proves nothing."[1]

IV.—Validity of Contracts.

It may be sought to invalidate a contract on the ground of the insanity of one of the parties thereto. To succeed, two things must be proved, viz.: (1) That the insanity existed at the time the contract was entered into, and (2) that by reason of insanity the contracting party was then mentally incapacitated to a certain extent or degree, viz.: that he was incapable of "understanding it, and of forming a rational judgment as to its effect upon his interests" (Indian Contracts Act [IX. of 1872], sect. 12). The law of England, however, makes certain exceptions to this general rule, viz.: (1) An insane is "liable for the price of necessaries, *i.e*, goods

[1] Judgment *in re Toplis*, Taylor, ii., 524.

suited to his rank and position, actually ordered and enjoyed by, and bonâ-fide supplied to him;"[1] and (2) an executed contract will not be invalidated, especially if the parties cannot altogether be restored to their original position, provided the contract is a fair and reasonable one, and the other party thereto had no reason to suppose the individual to be insane at the time of making it.[2] According to the law of England, marriage is a contract. Hence a marriage may be declared null and void on the ground of the insanity of one of the parties thereto at the time of entering into such contract. The degree of mental incapacity which must be proved in order to, *per se*, invalidate a marriage may be stated to be incapacity "to understand the nature of the contract and of the responsibilities and duties it creates" (see *D.* v. *D.*, *Case* D 39). Weakness of intellect coupled with undue influence, has been held to be good ground for invalidating a marriage (see *Case* D 40); hence the suitability, or otherwise of the marriage, may be one of the points for the consideration of the Court.

C.—IMPOSITION OF RESTRAINT.

When an individual by reason of unsoundness of mind is mentally incapacitated to a certain extent or degree, restraint may be lawfully imposed upon him. This restraint may be either immediate, or may be imposed (after certain conditions have been complied with), by relegation to an asylum. Hence in regard to the imposition of restraint, we have to consider three questions: (1) What degree of mental incapacity justifies the imposition of immediate restraint? (2) What degree of mental incapacity justifies the imposition of restraint by relegation of the individual to an asylum? and (3) What are the conditions which must be complied with, before an insane person is relegated to an asylum?

1. What degree of mental incapacity justifies the imposition of immediate restraint?

By the common law of England a person of unsound mind may be lawfully restrained from inflicting physical injury on himself or others. Restraint also, imposed in good faith on a person of unsound mind, for the purpose of preventing him from injuring himself, would come within the general exceptions of the Indian Penal Code, relating to acts done for a person's benefit (see sects. 89 and 92, *ante*, page 2). Probably, also these exceptions would be held to extend to such restraint as might be necessary to prevent an individual inflicting injury on others. Hence the degree of mental incapacity which, in a person of unsound mind, justifies the imposition of immediate restraint, is mental incapacity

[1] Browne, p. 7. [2] *Ibid.*, *Molton* v. *Camroux*, 4 Exch. 17.

to an extent which renders him dangerous to himself or others. Immediate restraint can, however, only be lawfully imposed, either (*a*) with the consent of the person having lawful charge of the insane individual; or (*b*) without such consent if the circumstances of the case are such, that the consent cannot be obtained in time to prevent danger. Further, immediate restraint thus imposed is only lawful so long as the danger exists. Imposition of immediate restraint is justifiable also, under similar conditions in cases of delirium from disease, *e.g.*, delirium tremens. In imposing immediate restraint in cases of delirium from disease, a medical man must recollect that from the nature of the case, the danger is liable to cease suddenly, and that restraint continued after danger has ceased, may be a ground of action against him.

2. What degree of mental incapacity justifies the imposition of restraint by relegation of the individual to an asylum?

By the law both of India and England, a medical man in relegating an insane person to an asylum, must certify that the individual is insane, and that he is "a proper person to be taken charge of and detained under care and treatment." Obviously a proper person to be detained under care in an asylum is one, who being insane [1] is dangerous to himself or others, and medico-legal writers are agreed, that this extends also to one who by reason of insanity is likely to injure his own property, or the property of others. Taylor [2] infers that relegation to an asylum simply for the purpose of treatment is not justifiable, but from the remarks of Lord Coleridge, C.J., in the case of *Neave* v. *Hatherley* (see *Case* D 41), it would appear that relegation to an asylum simply for the purposes of treatment is justifiable in cases, where the circumstances are such that efficient treatment cannot be employed unless the individual is so relegated. It may further be pointed out that when restraint by relegation to an asylum has been lawfully imposed, the responsibility for alleged unnecessary continuance of such restraint, no longer rests with the medical practitioner under whose certificate the restraint was originally imposed.

3. What are the conditions which must be complied with before an insane person is relegated to an asylum?

An insane person may be sent to an asylum by order of a Court, or he may be sent (in England) under the provisions of 16 & 17 Vict. cc. 96 & 97; or, in India, under the Lunatic Asylums Act (Act 36 of 1858).

[1] Not simply suffering from delirium of disease; this renders him a fit subject for a hospital, not for an asylum.

[2] Taylor, Manual, p. 709.

In England, by sect. 74, c 97, no person (not a pauper) may be received into an asylum without (1) an order from some person according to a certain specified form; and (2) the medical certificate also, according to a particular form, of two registered practitioners, not in partnership, and not one an assistant to the other, "each of whom shall separately from the other have personally examined the person to whom it (the certificate) relates, not more than seven clear days previously to the reception of such person into such asylum." In cases of emergency, however, a patient may be received on one medical certificate only, provided that two other medical certificates are furnished within three days. By sect. 12, c. 96, no medical practitioner who is interested in or attends a licensed house or hospital, or whose father, brother, son, partner, or assistant, is wholly or partly the proprietor of, or a regular professional attendant in such house or hospital, shall sign any certificate for the reception of a patient into it. By sect. 67, c. 97, a pauper lunatic may be received on one medical certificate only, if sent by order of a Justice; or under certain circumstances, if the certificate is accompanied by an order for the reception of the lunatic, signed by an officiating clergyman of, and the relieving officer or overseer of his parish.

In India, according to the Lunatic Asylums Act (36 of 1858), lunatics (other than lunatics sent to asylums by order of a criminal court) may only be received into an asylum under the following conditions:—

1. If sent by a magistrate, commissioner of police, or civil court. In this case only one medical certificate is required. This certificate must be in the form shown as Form A in the schedule of the Act;[1] and must be signed by a "medical officer"[2] after examination of the lunatic (sects. 4 and 8).

2. (But this applies to Lunatic Asylums in the Presidency towns, and stations of the Straits Settlements only.) On presentation of an order and statement filled up in the form shown as Form B in the schedule of the Act,[1] accompanied by "the medical certificate containing the particulars of Form A in the schedule to this Act (Act 36) of two persons, each of whom shall be a physician or surgeon, and one of whom shall be a Presidency surgeon, or a surgeon in the employment of the Government" (sect. 7).

Sect. 2 of Act 36 provides for the appointment of visitors to asylums, two or more of whom by sect. 3 are to visit the asylum at least once in every month; and by sect. 12, defective or incorrect orders or medical certificates may, with the sanction of two or more of the visitors, one of whom must be a medical officer,

[1] See Appendix XXVIII.

[2] Bomb. G. R. Genl. Dept. No. 4, of 3 Jan. 1883, directs that no medical officer of lower rank than an assistant-surgeon is to be called upon to examine lunatics under these sections.

"be amended by the person or persons signing the same." The form of medical certificate prescribed in the Indian Act (like that prescribed in England), requires the person signing it, it will be observed, to give the grounds on which his opinion has been formed, in the form of (1) facts indicating insanity observed by himself, and (2) other facts (if any) indicating insanity communicated to him by others.

Under head (2) must also be stated the name of the person from whom the information was received.

It may now be pointed out, that in examining a patient previous to filling up and signing such a certificate, a medical man is bound to exercise the greatest care. As remarked by Lord Coleridge (see *Case* D 41), his examination should be "a real inquiry, a real weighing and sifting of evidence, a real serious and solemn exercise of judgment." Negligence or want of care on his part (not simply an error in judgment) renders him (see *Hall* v. *Semple, Case* D 42) liable to be cast in damages, on an action being brought against him. Obviously, a medical man unless he has himself observed facts indicating insanity in the patient, is not justified in signing such a certificate. To rely solely on the statements of others in such a case, amounts to culpable negligence.

Further, the facts relied on and embodied in the certificate as facts indicating insanity, must be facts which really do so. Numerous instances are quoted by Taylor[1] on the authority of Dr. Millar, of certificates filled up with facts other than "good facts," or facts really indicating insanity. Some of these consist of mere statements of the existence of peculiarities of appearance or temper, not of themselves sufficient to show the existence of insanity; *e.g.*, Has an insane appearance, or, is violent in temper and very abusive, or refuses to take medicine. Others again are statements either to the effect, that the individual labours under delusions, without specifying precisely what these delusions are; or statements to the effect that the individual labours under a particular belief, such as from its nature may possibly be true, unaccompanied by any definite statement to the effect that such belief has been inquired into and found to be untrue. A fact to be a good fact really indicating insanity, must either clearly show the existence of a delusion, or the existence of such conduct as cannot be accounted for on the supposition of sanity.

[1] Taylor, ii., p. 512.

APPENDIX.

ILLUSTRATIVE CASES—PART A.

(See Chapters I. to VI.)

Signs of Death. Identity.

1. Cases of Suspended Animation. (a) Case of Colonel Townshend, quoted from Cheyne (Guy, p. 239):—"He (Colonel Townshend) told us that he had sent for us to give him some account of an odd sensation he had for some time observed and felt in himself, which was that, composing himself, he could die or expire when he pleased, and yet by an effort or somehow he could come to life again, which it seems he had sometimes tried before he had sent for us. We all three felt his pulse first; it was distinct though small and thready, and his heart had its usual beating. He composed himself on his back, and lay in a still posture some time; while I held his right hand, Dr. Baynard laid his hand on his heart, and Mr. Skrine held a clean looking-glass to his mouth. I found his pulse sink gradually, till at last I could not feel any by the most exact and nice touch. Dr. Baynard could not feel the least motion in his heart, nor Mr. Skrine discern the least soil of breath on the bright mirror he held to his mouth. Then each of us by turns examined his arm, heart, and breath, but could not by the nicest scrutiny discover the least symptom of life in him. This continued about half an hour. As we were going away (thinking him dead), we observed some motion about the body, and upon examination found his pulse and the motion of his heart gradually returning; he began to breathe gently and speak softly." Colonel Townshend died the same evening, and on post-mortem examination all the viscera were found healthy except the kidneys, for disease of which he had been long under treatment.—**(b) From Ogston, p. 364.**—Dr. Duncan, of Edinburgh, in his lectures used to mention the case of "a medical student who, like Colonel Townshend, simulated successfully the appearance of death; he died, however, some time afterwards of disease of the heart."—**(c) From Ogston, p. 365.**—Professor Maschka, of Prague, related in his lectures that "a mature child, which showed no signs of life, was placed in the anatomical rooms of the University, left there for fourteen

hours, and then taken to the physiology class-room. On laying open the chest no blood flowed from the integuments and soft parts in front. When the heart was reached, it was seen pulsating at the rate of twenty beats per minute. The lungs were seen to be in the fœtal condition."

2. Time of committal of murder indicated by condition of the body. Case of Gardner, the sweep (Taylor, i., p. 83).—Gardner lived with his wife and another woman, their servant. The wife was found dead in her bedroom, with wounds on her throat, at 8 A.M. Her extremities (which were uncovered) were cold, and rigidity was well marked. The circumstances of the case conclusively indicated murder, and Gardner was suspected and brought to trial. Gardner was proved to have left the house on the morning of the murder at 4 A.M., and was absent until after 8 A.M. The defence alleged that the woman was murdered during Gardner's absence. The medical witnesses in the case gave it as their opinion that, from the condition of the body when first seen at 8 A.M., it was clear that the woman had been dead more than four hours. Hence she must have died before Gardner left the house. This and the other evidence in the case led to the conviction of the prisoner.

3. Identity of dead bodies.—Convictions for murder.—Reappearance of the individual alleged to have been murdered. (a) From Chevers, p. 54.—Ibrahim Beg, a wealthy mahajun, was tried and convicted of the murder of his young wife Chumbelee. On the day previous to the one appointed for the execution of the convict, an individual informed a young English civilian that Chumbelee was alive, and led him to the place where she was kept concealed by a gang of fakeers in a subterranean chamber of a tomb. It then turned out that the whole affair was a conspiracy got up by a man named Khan Beg, the mahajun's next heir. This man, with the assistance of one of the mahajun's servants, first excited Ibrahim Beg's jealousy, and led him to beat his wife. Her loud screams were heard by the neighbours. They then abducted the wife and handed her over to the fakeers, in whose custody she was subsequently found. Just before the quarrel between Ibrahim Beg and his wife, the conspirators had got hold of a female body. They cut off the head of this, put on the arm of the corpse one of Chumbelee's bangles, and buried the body in the courtyard of Ibrahim Beg's house. Here it was discovered on the day after the quarrel, and was supposed to be the body of Chumbelee. The man who gave information that Chumbelee was still alive was a subordinate in the affair, who was dissatisfied with the remuneration given him for his services. —**(b) From Chevers, p. 69.**—In the Mirzapore district, a Rajpoot widow known to be unchaste eloped with a paramour. The headless body of a woman was found in a well, and was supposed to be the body of the widow who had eloped. The widow's brothers

were charged with her murder, confessed their guilt (?), and were convicted. Just before they were sentenced the missing widow appeared; she had heard by chance that her brothers were charged with killing her, and came forward to clear them. The brothers said they had confessed to the murder because they thought it was hopeless to plead innocence.

4. Identity of fragments of bodies. (a) **Dr. Parkman's case** (Guy, p. 32).—Dr. George Parkman, of Boston, U.S., was last seen alive entering the medical institution in which Dr. Webster was Lecturer on Chemistry. A week afterwards, suspicion having been excited, search was made in Dr. Webster's laboratory, and the remains of a human body discovered. In one place a pelvis, right thigh, and left leg, were found; and in another the entire trunk and the left thigh. Among the ashes in the furnace of the laboratory, fragments of bone, blocks of mineral teeth, and a quantity of gold, were also found. Some of the chief points in the case were: (1) No duplicate parts were found. (2) The pelvis was clearly that of a male. (3) The parts of the body were free from all traces of the preservative fluids always employed in the dissecting room of the college. (4) Piecing the parts together, it was estimated that the height of the body of which they formed portions was 70½ inches, or exactly the missing Dr. Parkman's height. (5) Three of the fragments of bone found in the ashes of the furnace when put together, made up the greater portion of the right half of a lower jaw. This was of peculiar shape, certain teeth were wanting from it, and it was found to fit exactly a cast of Dr. Parkman's jaw, which had a short time before been taken by a dentist who had supplied Dr. Parkman with artificial teeth, similar to those found in the furnace ashes. Dr. Webster was convicted, and subsequently confessed that he had murdered Dr. Parkman.—(b) **From Chevers, p. 66.**—A married woman and her child, a girl æt. four or five, disappeared at Meerut, under circumstances pointing to their having been murdered by a man named Kulloo, the woman's paramour. Kulloo absconded, but was subsequently apprehended while trying to sell ornaments proved to have belonged to the missing woman. Kulloo, when apprehended, made contradictory statements accusing other parties of the murder, and ultimately led the police to a place where several human bones were found, among them portions of two skulls, one small, the other larger. Among the fragments of the larger skull was an entire upper jaw (and half a lower jaw) with the teeth attached, and, near the bones, clothes, identified as having been worn by the child, were found. The civil assistant-surgeon to whom the bones were sent reported the bones to be those of two children, one about eight years, the other about eight months old. On this Kulloo was convicted of theft only. A re-inquiry was ordered; and at the trial held fourteen months afterwards, the civil surgeon was

called as a witness and examined. He apparently had kept no notes of the case, and had no recollection of having seen among the bones sent to him the portions of the larger skull above mentioned. His opinion as to the age of the individual to whom the larger bones belonged, seems to have been founded solely on the size of the bones. The judge held it to be not improbable that the civil surgeon had mistaken the bones of a small adult female for those of a boy of about eight years old, and convicted the prisoner of murder, sentencing him to transportation for life. Here, had the portions of the larger skull been examined, and a record kept of their appearance, and of the teeth in the portions of the jaws, the question whether they belonged to a child of eight or to an adult could probably have been determined without difficulty.

5. Identity of the living. (a) The Tichborne case (Guy, p. 686).—At the trial of this case in London, in 1874, the main question was whether an individual who had laid claim to large estates was or was not Roger Tichborne. Roger Tichborne was believed to have perished at sea twenty years previously. Some of the witnesses expressed their belief that the claimant was really Roger Tichborne; the majority, however, denied this. The following were some of the main points in the case:—(1) It was proved that Roger Tichborne had been bled repeatedly from the arms, and once also from the ankles and temple; also that he had tattoo marks on the left arm. None of these marks were present on the body of the claimant. (2) Comparison of the features of the claimant with a photographic portrait of the true Roger Tichborne showed the following differences: (*a*) The eyes of Roger Tichborne tended upwards from the nose, those of the claimant tended downwards; (*b*) the ears of the claimant were about one-third longer than those of Roger Tichborne; (*c*) the central groove joining the nose to the upper lip was much wider in Roger Tichborne than in the claimant. (3) The claimant was acquainted with many of the events in the life of the true Roger Tichborne, apparently picked up from various sources; he, however, (*a*) did not know the Christian names of Roger Tichborne's mother; and (*b*) could neither read nor speak French, although the true Roger Tichborne spoke French fluently.—**(b) The case of Martin Guerre** (Guy, p. 28).—In the second half of the sixteenth century, Martin Guerre, then a young man of twenty, absconded from his village in Languedoc, under fear of being charged with theft, leaving behind him his young wife and infant son. Martin Guerre, it was afterwards proved, enlisted as a soldier, and became extremely intimate with a comrade of bad character named Arnauld de Tilh. Eight years after Martin Guerre's disappearance from his home, Arnauld de Tilh appeared there, represented himself as Martin Guerre, and was at once accepted as the latter by all Martin Guérre's relatives,

including his wife. The impostor, mainly through his having become acquainted with all the true Martin Guerre's secrets, was able to carry on his imposture with success for several years. At the end of that period a quarrel arose between the impostor and Martin Guerre's uncle, when the latter denounced the former, who was put on his trial. At the trial, of 150 witnesses, forty swore that the accused was Martin Guerre, and fifty that he was not; the remaining sixty were in doubt. Martin Guerre's wife was quite satisfied that the accused was not an impostor. The trial resulted in the condemnation of the accused. He appealed. The Appeal Court found the evidence so extremely conflicting, that they were inclined to reverse the judgment of the lower Court, when the true Martin Guerre appeared. Arnauld de Tilh was thereupon condemned, and subsequently confessed his imposture. Some of the points in this case were: (1) The accused "had double eye-teeth in the upper jaw, a scar on the forehead, the nail of the left forefinger sunk in the flesh, and four warts on the right hand—all peculiarities possessed by the true Martin Guerre." In other personal peculiarities, however, the accused differed greatly from the true Martin Guerre. (2) "Martin was a skilled fencer, which Arnauld was not; and Arnauld could not speak even a few words of Martin's native Basque language."

6. Cases of doubtful sex. (a) Case of Levi Suydam (quoted by Woodman and Tidy, p. 678, from the 'American Journal of the Medical Sciences,' for July, 1847).—"In the spring of that year, Suydam presented himself first as a freeman, and then to vote in a contested election. Dr. Barry having found an imperforate penis with a depression in the site of the male meatus, a short urethra opening underneath the penis (hypospadias), and a cleft scrotum with a small but perfect testis in its right half, pronounced him to be a male and entitled to vote. Dr. Ticknor, who objected at first, came to the same conclusion. A few days after it was discovered, by his sister's and his own confession, that Suydam regularly menstruated, and had done so for years. His figure was feminine, the breasts were well developed; and on passing a sound into the urethra, instead of reaching the bladder it passed into a cavity like the vagina, three or four inches deep. It was also said that this urethra had been made for him by the accoucheur who attended at his birth."—**(b) Tidy's 'Legal Medicine,' vol. i. p. 353, case 128** (from 'Medical Gazette,' vol. xix. p. 135).—Æt. fifty-five at death. General configuration, that of a woman (during life celebrated anatomists had formed different opinions as to the sex). At the post-mortem there were found, on the right side, a withered testicle, a penis, and a prostate gland; and on the left an ovary, uterus, vagina, and Fallopian tube.—**(c) Case of Hubert I. Pierre** (from Beck's 'Medical Jurisprudence,' p. 70).—Died at the age of seventeen. No down on chin or lip. Breasts of middle size, with large areolæ.

The bust was feminine, but the hips were imperfectly developed. Penis four inches long, with prepuce, but imperforate, a little indentation occurring at the ordinary spot of passage. Under the penis was a fissure resembling that between the labia. The left labium contained a testicle with spermatic vessels, whilst in the right a body descended when the abdomen was pressed, which proved to be an imperfect uterus, with Fallopian tube and ovary. A rudimentary vagina one inch long was also present."—**(d) Case quoted by Ogston** (p. 52).—"A person affected with hypospadias was married for fifteen or twenty years, and during all that time was treated as a female. Sexual intercourse was regularly effected by the canal of the urethra, nor was it until the period just mentioned had elapsed, that it was discovered that the individual was a man."

Wounds.

7. Wounds self-inflicted in support of a false charge.—**(a) From Chevers** (p. 357).—"In 1853 three native women and two children were found lying dead in a heap with their throats cut in their bungalow at Baitool. The husband of one of the females gave the alarm, stating that the crime had been committed by dacoits (gang-robbers), who had also wounded and bound him! It, however, soon became evident that this man was the murderer." . . . His wounds were very slight, the chief one being on the thigh, about "three inches long, and in no part penetrating completely through the true skin;" the others were shallow scratches exactly parallel to the first, and the amount of blood on his clothes and body was much greater than could have flowed from his wounds.—(b) **Harvey's Report** (p. 117).—"A mussulmani at Dehra Ismail Khan, examined by Surgeon G. P. Mackenzie, in September 1872, had a slight cut scarcely skin deep, and two or three smaller scratches on throat. She accused her husband of attempting to murder her. The injuries were pronounced to be very trifling, and probably self-inflicted. She was convicted of bringing a false charge, and sentenced to six months' imprisonment."

8. Wounds inflicted by consent in support of false charges.—Chevers (page 358) states on the authority of Mr. Perceval that at one time two or three gangs existed in Bombay who cut and wounded each other for the purposes of extortion. "They used to cut one another's necks and arms by turns as the lot fell, and accuse some rich passer-by of having done it. . . . It fell to the lot of a youthful member of one of these gangs to have his neck cut. The person appointed to cut him was a drunken barber, who, instead of making a slight cut, inflicted a mortal wound. The gang fled, abandoning the youth, whose dying confession led to their arrest."

9. A similar case.—In a case tried before the High Court, Bombay (the Ahmedabad Conspiracy Case), the evidence went to show that certain individuals, A. B. and others, wishing to injure C. D. and others, proceeded as follows. They hired two men to wound a third, E., instructing E., after receiving the injury, to first of all accuse them (A. B. and others) of the assault, and then to make a pretended confession that this was a false charge brought at the instigation of C. D. and others. This programme was carried out, E. very nearly dying, owing to the severity of the wounds inflicted on him, and C. D. and others were convicted of instigating E. to bring a false charge against A. B. and others. After C. D. and others had suffered a considerable portion of their sentence of imprisonment the truth was discovered. A. and B. having brought a civil action for damages for malicious prosecution against C. D. and others, who were then in gaol, it appeared on the civil trial that A. B. and others had been instigated by an individual who had kept himself in the background, but was the real mover in the whole plot. A. and B. having succeeded in convicting C. and D., had caused an attorney's letter to be written to this individual demanding payment of the promised reward!! and it was principally by proof of this fact that the real truth came out.

10. Murder in support of a false charge.—*Reg.* v. *Muhammad Amanji and Husan Amanji* (Bo. H. C. Rep. vol. viii., 1871, p. 110).—A summary of the main facts in this case and two others connected with it (*Reg.* v. *Muhammad Valli*) and (*Reg.* v. *Alibhai Mitha*) is as follows. It appeared that two factions existed in the village of Karmar in the Broach Collectorate—A and B. Alibhai Mitha and Muhammad Amanji were members of faction A, and Muhammad Valli was a member of faction B. The two factions had a scuffle, in which one of the members of faction B got a blow on the head, and was taken into Broach. On this, faction A held a consultation, at which it was determined to break or bruise the head of one of their own party (Alibhai Mitha's old mother), and take her into Broach as a sort of make-weight against the broken head on the side of faction B. This was done apparently with the consent of the sufferer, and a false charge laid against faction B. While Alibhai's mother was in hospital, Alibhai's faction (faction A) held another consultation, the result of which was that they determined to poison Alibhai's mother in order to have a death on their side instead of simply a broken head. Accordingly they put arsenic into some food, gave it to the old woman, who thereupon was attacked by violent vomiting, which it was stated brought on rupture of the spleen, from which she died. On this, Muhammad Valli (a member of faction B) brought his sister out of his father's house and killed her by striking her on the head with an axe. She was heard just before she was struck to say, "Why do you kill

me for other people?" Muhammad Valli then dashed his own head violently against a wall—all this appears to have taken place in presence of the girl's father and other witnesses—and a false charge of murder and assault was then laid against faction A. Next Muhammad Amanji, a member of faction A, expressed his intention of killing himself as a set-off against the girl's death. On this his old mother begged that she might be killed instead. Thereupon Muhammad Amanji and his mother went into the back-yard of their house, and shortly afterwards the former rushed out, with a wound on his chest, calling for the police pâtil to come and take the deposition of his wounded mother. This was done, and the mother taken into Broach to the hospital. There her wounds were considered slight, and fifteen days after her admission she was discharged, and went back to her village. About six days afterwards her corpse was brought back to hospital. The civil surgeon certified that "these wounds did not bring about the death of this woman; she died of old age." This, however, the Court appeared to doubt. In giving judgment, Gibbs, J., remarked: "The evidence shows . . . that there are two factions in this village, and that murders have been committed on each side—not, as would be naturally expected, by members of one faction on a member of the other, but by members of one faction on a helpless female of their own, so as to throw either the guilt of blood or the blame of the crime on the other party. Such a state of things is hardly credible, but this is an instance of truth being stranger than fiction."

11. Circumstances under which wounds were inflicted inferred from position and form of blood spots.—In the case of Spicer (Taylor, i., p. 549), a woman was killed by a fall down a stair, fracturing her skull and vertebral column. A branch of the right temporal artery of the deceased was found divided, and on the wall at the top of the stair, on the right hand going up, several spots of blood were found, of the form and appearance of spots resulting from the spouting of a small artery. From the form and position of these spots, it was probable that deceased received a blow on the head while ascending the stair, and fell backwards to the bottom, the fall causing the injuries which resulted in her death.

12. Evidence from bullet marks.—(a) **From Taylor** (i., p. 689).—Several shots were maliciously fired into a church. Some of the bullets traversed a window, making holes in the glass, and struck against a wall on the other side of the church. A straight line from these two points reached a window on the opposite side of the street, from which it was afterwards ascertained that the shots had been fired.—(b) **From Woodman and Tidy** (p. 1091).—"Sir Astley Cooper, when called to see Mr. Blight, of Deptford, who had been mortally wounded by a pistol shot, inferred from an examination of the localities that the shot

must have been fired by a left-handed man. The only left-handed man on the premises at the time was a Mr. Patch, a particular friend of the deceased, who was not in the least suspected. This man was, however, subsequently tried and convicted of the crime, and made a full confession of his guilt before execution."

13. Fabrication of evidence of suicide (from Ogston's Lectures, p. 376).—"At the trial of a person named Saville, in 1844, it came out in evidence that the deceased, his wife, was found dead with her throat cut, and that there was a razor not grasped but lying loosely in her hand. There was no blood upon the hand which held the razor, and this, together with the fact of its being loose, rendered it certain that the weapon must have been placed there by some one after the woman's death."

14. Alleged fabrication of evidence of suicide (*ibid.*).—"In a case tried in France, in 1835, a man narrowly escaped conviction as the murderer of his father. The latter had been found dead in a sitting posture, with a recently-discharged pistol grasped in his right hand, the weapon resting upon the thigh in such a way that the slightest motion of the part would apparently have caused it to fall. It was assumed that the son had produced the injury to the face, which had been the cause of death, and had afterwards placed the pistol in his father's hand, in order to induce the supposition of suicide. The medical evidence, by showing that the grasping of the weapon could not have been simulated after death, led to an acquittal."

15. Attempt to conceal homicide by fabricating evidence of accident (Assistant-Surgeon Duncan, in McLeod's Report, p. 31).—The body of a man was found at the foot of a mango tree, with a bundle containing mangoes tied round the neck. On the body was: (1) Around the lower part of the neck, extending from right side of trachea to posterior edge of left sterno-mastoid muscle, a brown mark, as if from pressure of a cloth or large cord. (2) Abrasions and scratches with dust adhering, on right front of chest and anterior and outer aspect of right arm. (3) On right side of face and neck several bruises elongated in shape. (4) Bruises and abrasion over right parietal protuberance. Blood in large quantity extravasated under scalp. All right half of the skull broken into fragments, fissures radiate in all directions, all the fragments irregular in shape, and lying loose on the brain. An opinion was given that the injuries were caused by blows from a weapon, not by a fall from a tree. Subsequently one of the accused confessed that he, deceased, and several others, had been stealing mangoes, when a dispute arose as to the division of the plunder, and some one knocked deceased down with a latee. The body was then conveyed some distance (probably dragged along the ground) and placed under the tree, so that it might appear that a fall therefrom was the cause of death.

16. Locomotion after severe injuries (Ind. Med. Gaz., 1867, p. 200, Dr. Hutchinson).—An old man was severely beaten with a split bamboo: after the beating he walked to his house, a distance of about half a mile, and died almost immediately. On post-mortem examination, the seventh and eighth ribs on each side were found fractured, the spleen ruptured, and the right lobe of the liver all but divided transversely by a bifurcated rupture 8 inches long, and 1½ inch deep and broad.

17. Arrow wound (Ind. Med. Gaz., 1875, p. 257, Dr. S. I. Manook).—A Hindu female, aged fifty.—An arrow, having first passed through the fleshy portion of the right forearm, had penetrated the chest between the eighth and ninth ribs, and was sticking in the body. On opening the chest, the arrow was found to have passed through the diaphragm, having slightly cut the upper surface of the right lobe of the liver, pierced through the lower lobe of the right lung, and penetrated about an inch into the spine behind the heart and root of the lung. There was a large quantity of fluid and clotted blood to the right of the spine, but the heart was uninjured.

18. Post-mortem appearances indicative of more than one form of violence.—(a) From Woodman and Tidy (p. 1081). —A girl died while her father was chastising her for stealing. On examination many marks of blows were found on her back, arms, &c., and under some of them a good deal of blood was effused. The injuries seemed scarcely severe enough to account for death. On opening the stomach "it was found much inflamed, and lined with a white powder, which proved to be arsenic." The girl had taken arsenic fearing her father's anger; she died in slight convulsions.—**(b) From Bombay Analyser's records.**—The body of a young Hindu female was brought to the dispensary at Parola (Khandesh). It showed no signs of decomposition. On the back and shoulders were linear marks of recent bruises, and there were bruises attributed to kicks on the genitals. On internal examination, the spleen was found ruptured, apparently from a kick or blow. There was no sign of inflammation of the stomach or intestines. The hospital assistant in charge of the dispensary reported the case to be one of death from external violence. The magistrate investigating the case, learning that deceased was seen vomiting and purging before death, ordered the viscera to be sent for analysis. Arsenic was found in the contents of deceased's stomach, and in some matters vomited by her during life.—**(c) From the same.**—In a case from Sheagaon (Bombay), the body of a Hindu female was found in a well eight days after she had disappeared from her village. On analysis, about twelve grains of arsenious oxide was found in the contents of the stomach of the deceased. No account of the post-mortem appearances present was furnished to the Chemical Analyser's office.

19. Disappearance of ecchymosis (Harvey's Med. Leg. Rep., p. 26).—"Dr. C. M. Russell, of Gaiah, found not the slightest contusion or mark on any part of the body of a man whose ribs were fractured, and who died of apoplexy caused by direct violence to the head, proved by extensive extravasation into the right temporal muscle." *Ghi* had been rubbed into the body to remove swelling and discoloration, and had "succeeded perfectly."

20. Death caused by compression of the testicles (Chevers, p. 479, quoting Bellasis' reports, p. 234).—"Moobraek was capitally sentenced in the Konkan (Bombay Presidency) in 1845, for having murdered a man with whose wife he had an intrigue, by seizing him by the testicles, which he wrenched in the most violent manner." It was shown that the deceased was a man in rude health, that he was not suffering from any disease, and that after the violence described "his testicles (scrotum) were swollen to the size of a cocoa-nut."

21. Death from a kick on the vulva (Taylor, i., p. 678, from *Lancet*, 1846, p. 478).—A woman, æt. thirty-six, while in a stooping posture was kicked by her husband in the lower part of the abdomen, and died in about an hour from loss of blood. There was no injury to the vagina or uterus. There was a wound about 1 inch long and ¾-inch deep, situated at the edge of the vulva, extending from the pubes along the ramus. The left crus clitoridis was crushed throughout its length, and from this the fatal hæmorrhage had taken place.

Hanging, Strangulation, and Suffocation.

22. Suicidal hanging with a handkerchief; partial recovery after half an hour's suspension (quoted by Tidy from the *Lancet* of the 6th of July, 1844: Dr. Shearman).—"The man was cut down, apparently lifeless, after half an hour. The face and neck were found swollen, and a livid ecchymosed mark existed below the thyroid cartilage; the fingers were bent, and the hands clenched. The man was bled, and in a few minutes began to breathe. He rallied after a few hours. This was succeeded by great restlessness, and by convulsions. He afterwards became calm, and spoke several times, but suddenly died from exhaustion, nineteen hours after he had been cut down. (Death was due probably to the injury to the cerebral circulation)."

23. Murder by strangulation, with subsequent suspension of the body (a) (Chevers, p. 592).—A man, of Mymensing, having intrigued with a widow, and not giving her sufficient means for her support, she complained to the village Punchayet, who decided that both parties should be beaten. The man was seized by his father, and was struck several blows, but the woman managed to escape. The paramour, enraged at having been

summoned and beaten before the Punchayet, pursued her with three of his relatives. On coming up with her, they strangled her, and, hanging her body on a tree hard by, reported that she had committed suicide.—(b) **From Chevers** (p. 592).—The father and brothers of a girl, of Tipperah, finding her in company with a man with whom she was intriguing, seized the man, and, holding him down by the neck, arms, and legs, strangled him. They then hung up the corpse, and reported that he had destroyed himself.—(c) **From Chevers** (p. 593).—A Bogra woman was found hanging. Post-mortem examination showed clearly that strangulation by hand, and not by hanging, was the cause of death.

24. Murder by mechanical violence, with subsequent suspension of the body.—(a) (Chevers, p. 597). A man of Sylhet struck his wife with a piece of split bamboo about the body, for eating more than her share of pawn, until she died. He then hanged her body on a tree.—(b) **From Chevers** (p. 598). The wife of a man living in the 24 Pergunnahs, having a criminal intrigue with another Hindu, she and her paramour enticed the unfortunate man out of his house at night, killed him, or rendered him insensible (it would seem by severe blows), and suspended his body to a tree.—(c) **From Chevers** (p. 598). It appeared, in a trial at Cuttack, that a Hindu, charging another with theft, beat him to death. The man's body was afterwards found suspended, with marks of violence upon it, in such a position as to render it evident that he had not hanged himself.

24. (d) A man, probably trampled to death, body found hanging (Harvey's Beng. Med. Leg. Rep., p. 83).—A Hindu, aged about sixty. Post-mortem appearances—"Face livid, and slightly swollen, especially on right side, on which the body had been laid. The tongue was not swollen or bitten by the teeth. A bruise about 1½ inches in diameter on right side of the forehead. A *livid depressed mark*, about ¼ inch in diameter, round the neck and behind right ear. In front of the neck the mark was between os hyoides and thyroid cartilage. There was another depressed mark under the forehead. *Head.*—Considerable amount of coagulated blood in tissues of pericranium, corresponding to bruise on forehead. Brain healthy, slightly congested on surface. A considerable quantity of fluid in ventricles, and at base of brain. *Chest.*—A large quantity of extravasated coagulated blood among muscles and tissues covering the ribs. The ribs, from the third to the last, were fractured in two places on both sides; lungs healthy, uncongested; heart empty. *Abdomen.*—Extensive rupture of liver on its posterior aspect; all other organs healthy."

24. (e) Punctured wound mistaken for a gunshot wound, body suspended after death (Harvey, *ibid.* p. 214).—In this

case a mussulmani, aged thirty-eight, was at first reported to have died from hanging. There was a rope close under the chin, passing upwards behind the ears, and the head was bent on the chest. On lifting the head, a wound, described by the medical officer who made the examination as a gunshot wound, was found between the attachments of the sterno-mastoid muscle, a little above the clavicle. The wound contained a large clot of blood, and its edges were turned downwards and inwards. Apparently it was not seen until the head was lifted. The right lung was torn through from apex to base, and a circular hole, of the same size as the one in the neck, passed right through the liver. The right kidney was bruised, but not wounded. A large quantity of blood was found in the cavity of the thorax, and a large quantity also in the abdomen. Deeming the wound a gunshot wound, the medical officer, not being able to find the bullet, gave as his opinion that it had probably passed behind the kidney into the thigh. The woman's husband afterwards confessed to having killed his wife, by thrusting a "pointed perfectly circular solid bamboo" into her body. The body was afterwards hung up to avert suspicion.

25. Suicidal hanging, partial suspension only (Ogston, p. 534).—A young woman, who had been in a desponding state for some days, was found dead in bed one morning, sitting in a reclining posture with a ligature attached to the top frame of the bed. All that had been required to effect her purpose of suicide had been to lean back after the cord was fixed. Two young girls who were in bed with her had not been awakened by the transaction, so quietly must it have been effected.

26. Accidental hanging (*ibid.* p. 528).—During the breakfast hour at a cotton mill near Aberdeen, one of the men was toying with a female fellow-worker to whom he was attached, and in sport threw around her neck a loose leather strap suspended from the roof of the apartment. At this moment the machinery was set a-going, and the girl was drawn up to the roof by the strap, and suspended there for a few minutes before the engine could be stopped, too late for saving her life.

27. Death from hanging. Arsenic found in the viscera, ? self-administered.—In a case from Shahapur (Thana district), the body of a Mahar was found hanging to a tree outside a village. On examination, there was found an abrasion of the skin round the neck just below the chin, commencing from the thyroid cartilage, and extending backwards and upwards on both sides, with discoloration of the parts around. Both lungs were found gorged with blood, and the brain was congested. The mucous membrane of the stomach was red, and had yellow patches on it. On analysis, arsenic was found in the viscera, about four grains being present in the contents of the stomach. No information could be obtained in regard to this case, except that it

was supposed that the poison had been administered to the deceased in food.

28. Death from apnœa; finger-marks on the neck, probably caused by deceased himself (Chevers, p. 580).—A man was found dead in the house of a prostitute, who had been his mistress for three years. Deceased's cousin was sleeping in the same house with another prostitute, and was called early in the morning by deceased's mistress, who appeared to be much alarmed at his (deceased's) condition. The cousin found him apparently dying, and removed him to his own house. A post-mortem examination was made, and showed death to be due to apnœa. Three marks were found on deceased's throat, such as would be produced by finger-nails. The surgeon who made the examination thereupon reported strangulation to be the cause of death, and deceased's mistress was tried and convicted of the murder. It, however, appeared that deceased had been subject to epileptic fits, and, many of the circumstances of the case being strongly against the supposition of homicide, the High Court reversed the conviction, holding that the marks on the throat might have been produced by deceased himself.

29. Accidental strangulation (Guy, p. 289).—Elizabeth Kenchan, an extremely dissipated, drunken, and disorderly woman, went to bed intoxicated with her bonnet on, and in the morning was found strangled in its strings. She had fallen out of bed, her bonnet became fixed between the bedstead and the wall, and she, being too drunk to loosen the strings, was strangled.

30. Accidental strangulation (Ogston, p. 543).—A man was carried to bed very drunk, and left there with his clothes on. It was supposed that afterwards he had got up so far as to lean over the front of his bed to vomit, with his hands pressed on the pit of his stomach, as he was found dead in this posture in the morning. His neckerchief was so tight around his neck, that the contraction thus caused would have sufficed to produce strangulation, from his inability to change his position, in the helpless condition he was in at the time. The inspection, by presenting all the appearances which were to have been expected under such circumstances, left no doubt but that accidental strangulation was the cause of death.

31. Accidental strangulation (Taylor's Principles, ii., p. 67).—A girl was accidentally strangled in the following manner. She was employed in carrying fish in a basket at her back, supported by a leather strap passing round the front of her neck above her shoulders. She was found dead sitting on a stone wall; the basket had slipped off, probably while she was resting, and had thus raised the strap, which had firmly compressed the wind-pipe.

32. Accidental suffocation (Chevers, p. 619).—"In 1859,

Dr. Whyte reported the case of a strong Madras Bheestie, into whose mouth a fish had jumped while he was bathing. On opening the mouth, the tail of a large cat-fish presented itself, with the body firmly fixed within the fauces, and filling up the isthmus completely. It had entered flat, so that the fin of one side was posterior to the velum, and opened out on any attempt being made to withdraw the fish. The operation of œsophagotomy was commenced and was abandoned. A piece of cane was made into a probang, and, with it, attempts were made to press the fish downwards into the œsophagus. It did pass downwards, when the patient at once ceased to breathe, gave one convulsive struggle, and died to all appearance. The trachea was immediately opened, and respiration was restored. In the course of the night the man coughed up the fish, the fins having become softened by decomposition."

33. Accidental suffocation (Chevers, p. 619).—"In 1865, a native boy about four years old was brought to the Calcutta Medical College Hospital, with a *coir* fish impacted in his glottis. These fish are very tenacious of life out of water. The poor child appears to have taken up the fish, and to have put its head into his mouth. In its struggles, its head passed the glottis, and all attempts to withdraw it were prevented by the catching of its gill-plates, anchor-wise, below the vocal chords. The child was suffocated."

34. Accidental suffocation (Chevers, p. 618).—"A private soldier, aged twenty-eight, was discovered, at night, by the man lying next to him to be breathing loudly and with great difficulty, as if there were some obstruction about the lower part of the trachea. He was at once removed to the dispensary, where he died in about fifteen minutes. Several small pieces of potato were found in both bronchial tubes, where these subdivided into small branches. There was great œdema of the glottis, no doubt from the irritation caused by a foreign body. The deceased had drunk some beer and also rum in the course of the evening, before retiring to rest. He had been sick, and had vomited while in bed."

35. Homicidal suffocation (Chevers, p. 166).—A very young man of Mirzapore was sentenced to death for murdering a boy of twelve for his ornaments, by filling his mouth with mud. The civil surgeon deposed that the cause of death was, in his opinion, suffocation and consequent congestion of the brain, produced by the mouth and air-passages of the deceased having been filled with mud, whilst he was alive.

36. Homicidal suffocation (Chevers, p. 616).—A lad from thirteen to fifteen years of age was sentenced, at Agra, to transportation for life for having robbed a girl of four, his near relative, of her ornaments, after having filled her mouth with *bhoosa*. The civil surgeon deposed that death had been caused

by suffocation, consequent upon the mouth of the deceased being filled with *bhoosa*.

37 (a). Homicidal suffocation (Chevers, p. 616).—An old woman of Tirhoot, finding a little girl of six digging up some grain from her field, felled the child with a heavy clod, and then suffocated her by pressing her clothes against her mouth, until she ceased to breathe. She then stripped off the clothes and ornaments, and buried the corpse.

37 (b). Homicidal suffocation (Dr. McReddie, in McLeod's Bengal Med. Leg. Rep., 1868–69, p. 36).—Post-mortem examination of the body of a girl aged about twelve. Body far advanced in decomposition; no marks of violence externally, but on cutting into the skin of the chest, extensive bruises and bloody effusions were found over the whole front aspect of the ribs. The ribs were not fractured. The right lung was natural, but the left had been most severely injured by compression and had become a jelly-like mass. The girl had probably been thrown down, and then had her chest compressed by the weight of her assailant's body.

Burns.

38. False charge of burning (Chevers, p. 532).—"In March, 1865, the assistant magistrate of Howrah sent me a girl about ten years old, for my opinion as to how certain marks on her cheeks, arms, and back were caused. She asserted that they were burned with a hot *chillum* (tobacco-pipe), whereas the accused declared that they had been made with some paint. I found a large circular brown mark on either cheek; each of these marks had a clean and perfectly-defined edge. The marks on the arms and back were parallel brown streaks, with clean edges; there was no vesication, but the cuticle was beginning to separate. Such even, clear-edged, symmetrical marks could not have been inflicted with a heated body upon any person who was not in a state of complete insensibility, and from their shape it was evident that they could not have been caused by the application of a *chillum*. I gave it as my opinion that a fluid irritant had been applied, and that the case had been trumped up."

39. Supposed attempt to conceal murder, by burning the body (Bombay Chemical Analyser's Report for 1883, p. 9).—In a case forwarded from Sukkur (Sind) some fragments of partly-burnt bones were sent for opinion as to whether the same were or were not fragments of human bones. Several of the fragments forwarded were clearly identified as fragments of the bones of an adult human being. A summary of the history of this case is as follows:—Two men started out together, one carrying an axe; after a time one of the two returned, the other seemingly having disappeared. Trackers were placed on the trail made by the two men, and they, on following the trail, came to a place where the

double trail ended, and a return single trail began: at this place the fragments of bone sent for examination were found.

40. Apparent wounds caused by burning (Taylor, vol. i. p. 715).—A boy, æt. two, was brought to hospital severely burnt, and died in three-quarters of an hour. There were gaping wounds on both knees. On the right side, a fissure in the skin commenced about the middle of the thigh, and proceeded for two inches and three-quarters to the inside of the patella, or knee-pan, where it became somewhat jagged, and making a sudden turn inwards, passed to the extent of two inches towards the back of the joint. A transverse laceration of the skin, three-quarters of an inch in length, was observed on the front of the left thigh, a little above the left knee; and another, which was also transverse and measured an inch and a half, was situated below, on the inner side of the joint. These fissures in the charred skin were all about three lines in width and two in depth, and exposed the fatty tissue beneath, which was white, and free from any effusion of blood. The edges of these fissures were not uneven, but they did not present the clean and smooth appearance usually observed in incised wounds. In several places some small vessels containing blood were observed running across the fissures; these, being more tenacious than the fatty tissue, had not yielded with it. From the absence of any trace of effusion of blood, the sound condition of the exposed adipose tissue, its exemption from the action of the fire, and the irregular character and appearance of the fissures, Mr. Curling considered them to have been occasioned by the influence of heat.

41. Case of so-called spontaneous combustion of the human body (Woodman and Tidy, p. 1010).—In the Philosoph. Trans., vol. xliii., p. 463, it is recorded that "Grace Pett, the wife of a fishmonger of St. Clement's, Ipswich, *used to go downstairs every night, half-dressed, to smoke a pipe.* On the 9th of April, 1744, she got up from bed as usual. Her daughter, who slept with her, did not perceive that her mother was absent till next morning when she awoke. Soon after this she put on her clothes, and, going down into the kitchen, found her mother stretched out on her right side, with her head near the grate. The body was extended on the hearth, with the legs on the deal floor, and it had the appearance of a log of wood consumed by a fire without apparent flame. On beholding the spectacle, the girl ran in great haste and poured some water over her mother's body, to extinguish the fire. The fœtid odour and smoke which exhaled from the body almost suffocated some of the neighbours, who had hastened to the girl's assistance. The trunk was in some measure incinerated, and resembled a heap of coals covered with white ashes. The head, the arms, the legs, and the thighs had also participated in the burning. This woman, it is said, *had drunk a large quantity of spirituous*

liquor, in consequence of being overjoyed to hear that one of her daughters had returned from Gibraltar. There was no fire in the grate, and the candle had burnt entirely out in the socket of the candlestick, which was close to her. There were also found near the consumed body, the clothes of a child, and a paper screen, which had sustained no injury. Her dress consisted of a cotton gown.

42. Spontaneous combustion put forward as a defence to a charge of murder (Taylor, Manual, p. 348).—"In March, 1850, a man named *Stauff* was tried at Darmstadt for the murder of the *Countess of Goerlitz*. He had assaulted the deceased in her chamber, and then set fire to the furniture, with a view to conceal his crime. The body and dress were partially consumed. As the means by which the fire was applied were not at once apparent, and the assassin had locked the doors of the room, some medical men took up the theory that the deceased had died from spontaneous combustion. The facts of the case were referred to Professors Liebig and Bischoff, of Giessen, and their report was issued in March 1850, at which date the man Stauff was put on his trial. They found no difficulty in concluding that a murder had been perpetrated, and the body wilfully *burnt after death*, for the purpose of concealing the crime." Stauff was convicted, and subsequently confessed that he had strangled the Countess, and then, heaping articles of furniture around the body, had set fire to them, with the object of concealing the murder.

Starvation.

43. Prolonged sleep (Taylor, i., p. 43).—A man of healthy habits, forty-three years of age, was at intervals subject to attacks of long and persistent sleep. He would retire to bed at his usual hour, and, without any warning symptoms, suddenly and almost immediately fall into a profound sleep, from which all the usual means would fail to arouse him. In this state his face and ears were pale; the skin was pale, and generally warm, but his feet were cold and livid, and the limbs quite relaxed. His pulse was soft, slow, and feeble; his respirations almost imperceptible, amounting to about eight or nine in a minute. He appeared like a person in a refreshing, tranquil slumber. There was no stertor or snoring. The longest period he ever passed in profound sleep was five days and five nights. He frequently slept three days, and occasionally four days, without waking, but his average period was two days. His secretions were suppressed, and no food was required. He commonly awoke suddenly, had no consciousness of the lapse of time, and retained a good remembrance of the last occurrences before he fell into this state. He had no dreams.

44. Homicidal starvation.—Death from disease set up as

a defence (*Reg.* v. *Staunton*, Taylor's 'Manual,' p. 469). Deceased, Harriet Staunton, had been kept in close confinement by the accused. She was seen, a few hours before her death, by a medical man, and was then insensible and collapsed. She died in a state of complete exhaustion. On post-mortem examination appearances indicative of death from starvation were found, the body-weight being only 74 lbs. instead of about 124 lbs., as it would have been in a healthy adult of the same age. The following post-mortem appearances of disease were present:—1. A slight tubercular deposit at the apex of the left lung. 2. A congested appearance of the cardiac extremity of the stomach, as well as of the duodenum. 3. Two small patches of miliary tubercular deposit (recent) upon the arachnoid, on the upper surface of the left cerebral hemisphere. There was no trace of meningitis, or of disease of the brain. In this case the defence was set up that death was the result of disease. The medical men who examined the body were of opinion that death was due to starvation. Three of the four accused were convicted.

45. Starvation; rapid gain of weight under proper feeding (Tidy, i., p. 603; *Lancet*, August 14, 1880).—Prisoner charged with starving her servant, æt. thirteen and a half years. The girl weighed thirty-five pounds. She suffered, in the cold weather, from chilblains and sloughing of the toes. When removed and properly fed she recovered her health, and gained weight at the rate of five ounces per diem for 129 days.

46. Pretended fasting.—Case of Ann Moore (Hammond's 'Spiritualism and Nervous Derangements,' p. 268).—About sixty-five years ago a woman of Sudbury, in Staffordshire, England, named Ann Moore, declared that she did not eat, and a number of persons volunteered to watch her, in order to ascertain whether or not she was speaking the truth. The watch was continued for three weeks, and then the watchers, as in other instances, reported that Ann Moore's was a real case of abstinence from food of all kinds. The Bible was always kept open on Ann's bed. Her emaciation was so extreme that it was said her vertebral column could be felt through the abdominal walls. This sad condition was asserted to have been caused by her washing the linen of a person affected with ulcers. From that time she experienced a dislike for food, and even nausea at the sight or mention of it. As soon as the watchers reported in favour of the genuineness of Ann's pretensions, her notoriety increased, and visitors came from all parts of the country, leaving donations to the extent of £250 in the course of two years. Doubts, however, again arose, and, bold from the immunity she had experienced from the first investigation, Ann, in an evil moment for the continuance of her fraud, consented to a second watching. This committee was composed of notable persons, among them being Sir Oswald Mosley, Bart., Rev. Legh Richmond, Dr. Fox and his son, and

many other gentlemen of the county. Two of them were always in her room, night and day. At the suggestion of Mr. Francis Fox, the bedstead, bedding, and the woman in it, were placed on a weighing-machine, and thus it was ascertained that she regularly lost weight daily. At the expiration of the ninth day of this strict watching, Dr. Fox found her evidently sinking, and told her she would soon die unless she took food. After a little prevarication, the woman signed a written confession that she was an impostor, and had "occasionally taken sustenance for the last six years." She also stated that during the first watch of three weeks her daughter had contrived, when washing her face, to feed her every morning, by using towels made very wet with gravy, milk, or strong arrowroot gruel, and had also conveyed food from mouth to mouth in kissing her, which, it is presumed, she did very often.

47. Pretended fasting.—Case of Sarah Jacobs, the Welsh fasting-girl (Taylor's 'Manual, p. 472).—This girl, æt. thirteen, was stated to have voluntarily abstained from any kind of food for a period of *two years*. She had kept her bed during that time, lying in it decorated as a bride, visited by hundreds of persons; in fact, she was thus publicly exhibited by her parents as a girl of miraculous powers. Her lips were moistened with water once a fortnight, but, according to the parents, no food was given to her. Four professional nurses from Guy's Hospital were set to watch the girl, and the result was, that after passing through the usual stages of actual starvation, she died on the ninth day! She refused to take food at any time during the strict watching, and voluntarily accepted a lingering death, rather than reveal the imposture. Her parents, and those around her, allowed her to die! An inquest was held, and a post-mortem examination presented the following appearances. The body was plump and well-formed, the membranes of the brain were much injected, the brain itself was healthy, and of proper consistency. There was a layer of fat, from half an inch to an inch thick, beneath the skin of the chest and abdomen. The contents of the chest were healthy. The stomach contained three teaspoonfuls of a semi-gelatinous substance, of the consistency of syrup, having a slightly acid reaction. The small intestines were empty, and presented no attenuation or thinning of the coats. In the colon and rectum there was half a pound of solid excrement, in a hardened state, which might have been there, according to the medical witness, a fortnight or longer. The liver was healthy, and the gall-bladder was greatly distended with bile; the kidneys and spleen were healthy, and the urinary bladder was empty. The parents were convicted of causing the death of their child by criminal negligence.

ILLUSTRATIVE CASES—PART B.

(See Chapters VII. to XXV.)

Poisons.

1. **Remittent form of opium poisoning** (Taylor on Poisons, p. 552, case of the Hon. Mrs. Anson).—"This lady swallowed, while fasting, an ounce and a half of laudanum by mistake. In a quarter of an hour emetics were given, but she did not vomit for half an hour, and she was not treated medically for two hours and a half. The matter then drawn from the stomach had no smell of laudanum. She was quite unconscious, and had lost the power of swallowing. After remaining in this comatose state for upwards of nine hours, the patient revived, her face became natural, the pulse steady, the power of swallowing returned, she was able to recognise her daughters, and in a thick voice to give an account of the mistake she had made. This state lasted about five minutes; the torpor then returned, she again sank into profound coma, and died in fourteen hours after the poison had been taken."

2. **One only, of a number present at a feast, killed by poison** (Bombay Chemical Analyser's Report, 1880-81).—In a case which occurred in Poona, a man was reported to have died six or seven hours after partaking of food at a feast with about one hundred and twenty-five other persons. No complaint was made by his relations, and the body was buried. Some days afterwards, an anonymous writing was found outside the magistrate's court, stating that deceased had been poisoned, and an inquiry was ordered. It then turned out that deceased being of a different caste to the other persons present at the feast, was served with food separately from the rest, by a separate person, and that before death he suffered from symptoms of irritant poisoning. The body was then (eleven days after death) exhumed, and the viscera forwarded for analysis, when about twenty grains of arsenious oxide was found in the contents of deceased's stomach.

3. **Homicidal poisoning by colchicum** (*R.* v. *Catherine Wilson.* Taylor on Poisons, p. 512).—The prisoner was tried and convicted of the murder of a Mrs. Soames, who six years previously had died suddenly while being nursed by her. It was proved that, besides Mrs. Soames, three other persons had died suddenly after the administration to them by the prisoner

of food or medicine. In all four cases the symptoms were similar in character, viz., burning pain in the throat and stomach, intense thirst, violent vomiting and purging, collapse, and death from exhaustion without convulsions or loss of consciousness. In each of the four cases, also, the symptoms came on suddenly while the affected individuals were in a state of health, and in each case the death of the individual affected enabled the prisoner to acquire money or property. In each case the body of the deceased individual was exhumed, in one case within two months, in another about one and a half years, and in the other two six years, after death, but in all four cases no poison could be detected on analysis of the viscera.

4. Sudden death from swallowing, while heated, a quantity of cold fluid (Christison on Poisons, p. 120).—"A young man having just sat down panting, and bathed in sweat, after a severe match at tennis, drank greedily from a pitcher of water fresh drawn from a neighbouring pump. Suddenly he laid his hand on his stomach, bent forward, became pale, breathed laboriously, and in a few minutes expired."

5. Another case; death on the fifth day (*ib.* p. 121).—A soldier, after a hurried journey on a hot day, swallowed a quantity of iced beer. Six hours afterwards, shivering set in, followed by vomiting, anxiety, thirst, and frequency of the pulse. This was followed by great prostration, hiccough, and lividity of face. Death took place on the fifth day. On post-mortem examination, the mucous membrane of the stomach was found much reddened, and spots of extravasation were present. The stomach contained blackish matter, similar to what had been vomited during life.

6. Partial rupture of the stomach; symptoms similar to those of irritant poisoning (*ib.* p. 118).—A boy æt. fourteen, after eating and drinking heartily at a feast, was attacked with violent vomiting and purging. Next morning he was unable to swallow, his pulse became irregular, and pressure on the heart or stomach caused excruciating agony. These symptoms continued, and on the following day, after having vomited at intervals altogether about two pounds of blood, the boy died. On post-mortem examination, the inner coat of the stomach was found torn in many places, and that of the duodenum lacerated almost completely round.

7. Suicidal poisoning by sulphuric acid; appearance of urgent symptoms delayed (Taylor on Poisons, p. 183).—A man, æt. fifty-six, swallowed by mistake a dessert-spoonful of oil of vitriol. On admission into hospital, he was able to walk upstairs. He vomited slightly at first, did not appear very ill, had one brown fluid motion. The lining membrane of the mouth was brown. There were no urgent symptoms, but the patient died suddenly on the fourth day.

8. Another case.—"A girl, having swallowed a quantity of

concentrated sulphuric acid, sat quietly down to tea with some friends, although the quantity of acid taken was sufficient to cause death in a few hours."

9. Cases of Arsenical Poisoning; irritant symptoms slight.—Of three hundred and five fatal cases reported to the Bombay Chemical Analyser's Office during the ten years ending 1884, six (and as only meagre reports were furnished with many cases, possibly others also) were cases of this form. In four of the six the duration of the case was stated, and in all four death occurred within fourteen hours. In two of the six, there was no redness of the mucous membrane of the stomach. In one of these (a′), a female, who vomited once only, and had no purging, there was also no redness of the mucous membrane of the intestines. In the other (b′), a man, who had vomiting, but no purging, there was one patch of redness about the middle of the rectum. Post-mortem appearances of irritation were well marked in the other four cases. The symptoms reported in these were respectively: (c′) heat in the abdomen and thirst, slight vomiting and purging before admission into hospital, none after. (d′) Had vomited and purged four times before admission into hospital. While in hospital was drowsy, vomited once only, and had no purging. Conjunctivæ eight hours after admission noticed to be tremendously injected. (e′) Fever and severe pain in the abdomen, no vomiting, and no purging. (f′) Great thirst, restlessness, picking at the bed-clothes, and incoherence, no vomiting and no purging. Cases (*d′*) and (*e′*), although under medical observation, were not, during life, recognised as cases of poisoning. In case (*d′*) only one and a half grains of arsenious oxide was found in the contents of the stomach. In case (*f′*) the quantity found was one hundred grains. In the other four cases, the quantity found ranged from nine to fifteen grains.—Kenneth McLeod (Bengal Medico-legal Report for 1868-69) mentions a case where a girl, æt. seven, died in three hours, neither vomiting nor purging being present. Similar cases are recorded by various authors.—Christison (on Poisons, p. 308 *et seq.*, 1845) gives fourteen cases, all fatal within twelve hours, in which only slight irritant symptoms were present. In five of the fourteen, post-mortem appearances of irritation were either altogether absent, or trifling only.

10. Cases of Arsenical Poisoning; appearance of symptoms delayed.—(a) (Christison on Poisons, p. 299). A man took seven drachms of arsenious oxide at eight in the evening, went to bed at half-past nine, and slept till eleven, when he awoke with slight pain in the stomach, vomiting, and cold sweats —he died in nine hours.—(b) (Harvey, Bengal Medico-legal Report for 1870–72). Five persons, members of the same family, were poisoned by sweetmeat containing arsenious oxide; one of the five, a child æt. four, was roused from sleep to partake of the

sweetmeat and fell asleep again afterwards; in her case, the symptoms did not appear for two and a half hours, while in the other four—all adults—the symptoms appeared in about an hour. Two of the adults had not supped previous to eating the sweetmeat —both these died. The other three individuals had just finished their evening meal, and all three recovered.—(c) (Woodman and Tidy, p. 163). A female took half an ounce of arsenious oxide after a meal. No symptoms appeared for eight hours, when pain, vomiting, and purging set in; the stomach-pump was used. Result, recovery.—(d) (Christison on Poisons, p. 308, one of the fourteen cases referred to under 9). "The subject was a man so addicted to drinking, that his daily allowance was a pint of brandy. When first seen, there was so much tranquillity, that doubts were entertained whether arsenic had really been swallowed, but at length he was discovered actually chewing it. This state continued for nearly five hours, when some vomiting ensued. Coldness of the extremities and spasmodic flexion of the legs soon followed; and in a few minutes more, he expired."—(e) (*ib.* p. 309). A man swallowed three drachms of arsenious oxide, then went about for two hours, bidding adieu to his friends; he was then persuaded to take emetics, which caused free and easy vomiting; he hardly suffered at all for five hours, but died nine hours after taking the poison.

11. Perforation of the Stomach in arsenical poisoning.—This was reported in two of the three hundred and five Bombay fatal cases. (a′) In a case where a man died eight days after swallowing the poison: on post-mortem examination, the stomach was found inflamed and perforated at one place. In this case, however, the body had been buried, and was not exhumed for examination until twenty days after death. (b′) In a case reported by Dr. R. H. Batty: in this case a woman æt. about thirty-five, after a quarrel with her husband, swallowed a quantity of arsenious oxide. Duration of case not stated. Post-mortem appearances: much congestion of the membranes of the brain, also of the brain, lungs, kidneys, and liver. Heart normal. Œsophagus pale. Mucous membrane of stomach intensely red, with white particles adhering to it. Stomach perforated in three places on the posterior wall; two of the perforations very minute, the third "about the size of an ordinary quill;" from the latter, a long ropy mucous substance was protruding, in which there was a large quantity of minute white particles of arsenious oxide. Small intestines very red throughout, and containing particles of solid arsenious oxide.

12. Arsenical Poisoning; absence of usual appearances of inflammation of the stomach and intestines.—Christison, besides the five cases mentioned under 9, gives two other cases in which, on post-mortem examination, the mucous membrane of the stomach and intestines was found to be free from signs of

inflammation.—Besides cases 9 *a'* and *b'*; I have met with one other case (out of three hundred and five), in which only trifling signs of inflammatory action were present.—Harvey (Bengal Medico-legal Report for 1870–72) records absence of signs of inflammation in four cases out of one hundred and ninety-one. In one of these cases, the symptoms were "vomiting, purging, dryness of the mouth, thirst, anxiety, vertigo, and prostration." Death occurred in nine hours. In this case, the "whole of the intestinal tract" was found healthy.

13. Arsenical Poisoning; exceptional case of recovery ('Indian Medical Gazette,' 1872, p. 183).—A Parsee, admitted into the Jamsetjee Jejeebhoy Hospital, Bombay, had swallowed two masses of arsenious oxide. Sixteen hours afterwards, he passed, per rectum, a mass of arsenious oxide weighing eighty grains, and about forty-five hours after swallowing the poison he, in the same way, passed a smaller mass weighing twenty-five grains. The symptoms present were comparatively slight: there was no vomiting, but some diarrhœa; he was drowsy, his eyes were suffused, and he complained of headache and pain in the abdomen. He left the hospital quite well.

14. Arsenical Poisoning. Sweetmeat, &c., containing arsenious oxide in large proportion.—(a) (Bo. Chem. Analyser's Rep., 1872-73). In a case tried before the High Court, Bombay, it was proved that a man went up to another, a police Sepoy, while standing on duty in the public streets, and offered him some sweetmeat. He took it, bit off a mouthful, but finding it to have a gritty taste, spat it out. From eight hundred and sixty grains of the remainder, I extracted six hundred and ten grains of arsenious oxide. The police Sepoy, it was stated, had run away with the wife of the man who attempted to poison him.—(b) (Report for 1874-75). In a case from Ahmednagar, in which a man died in twelve hours after eating some poisoned bread, 5½ lbs. of the bread were found to contain one thousand five hundred and fifteen grains of arsenious oxide.—(c) (Report for 1878-79). A woman was seen feeding two children, æt. respectively four and six, with sweetmeat; both children died. The stomach of the elder child contained one hundred grains of arsenious oxide, and arsenious oxide was found in relatively large quantity in an unconsumed portion of the sweetmeat. The motive in this case was stated to be a quarrel with the parents of the children.—(d) (Report for 1882-83). Five persons, two adults and three children, inmates of a leper hospital in Bombay, were poisoned by sweetmeat containing arsenious oxide to the extent of about twelve grains per ounce. A large quantity of tamarinds was given at the same time to the same persons. The tamarinds contained about fifty grains per lb. of arsenious oxide. Some of the fragments of arsenious oxide contained in the tamarinds were as large as small beans. The motive in

this case, it was stated, was to get rid of a leper husband, whose wife the accused wanted to marry.

15. Arsenical Poisoning. Homicidal multiple cases (Bo. Chem. Analyser's Rep. 1880–81).—(a) In a case from Uran, a man and his wife and two children were all four poisoned by arsenious oxide contained in bread, given to them, it was alleged, by a neighbour who had a quarrel with the family. Two of the four died.—(b) In a case from Hyderabad (Sind), three children were poisoned by sweetmeat containing arsenic, given to them, it was alleged, by a man at enmity with their parents. One of the three, a baby, appears to have been poisoned by sucking the fingers of the other children.—(c) In a case from Ahmednagar, four persons, one of whom died, were poisoned by arsenious oxide contained in food. In this case, the accused was, it was said, on bad terms with the persons poisoned, and was seen seated near some cooking-pots in which their food was being prepared, leaving just as they were about to commence their meal.—(Report for 1879-80.) (d) In a case also from Ahmednagar, two children, both of whom died, and three adults were poisoned. In this case, it was alleged that the aunt of the children was the poisoner. She was seen, whilst the food was being cooked, of which the persons poisoned afterwards partook, to throw something into the pot containing it, and was known to have recently purchased arsenic.—(e) In a case from Hubli, it was reported that a man took some flour from a woman who was grinding it, afterwards throwing it back on to the handmill she was using. The woman having cooked the flour, she and her family partook of it: all were attacked with symptoms of irritant poisoning; all, however, recovered.—(f) (Report for 1881–82). In a case from Kaira, a man, it was alleged, gave some sweetmeat to another, with a request that he would give some of it to others that he named, saying it was a "*prasad*" (offering) of a goddess. The man to whom the sweetmeat was given, retaining some for himself, distributed the rest to the persons named, and these again gave some of it to three other persons. All who partook of the sweetmeat suffered from the usual symptoms of irritant poisoning; two died, one on the third, the other on the sixth day.—(g) (Report for 1879-80). At a mosque in Bombay, two Syuds were accosted by a man, afterwards arrested, who handed to them some food of which they partook; both were shortly afterwards attacked with symptoms of irritant poisoning. One of the two died; arsenious oxide was found in his viscera, and in the food which had been given to the two men. The individual arrested proved to be an imbecile, and apparently had no motive for injuring the persons poisoned. His story, which was very likely true, was that he was accosted by a man whom he did not know, and asked to take some food to the two Syuds.

16. Arsenical Poisoning; exceptional cases.—(a) (Bo. Anal.

Rep. for 1884.) In a case tried before the High Court, Bombay, four men were charged with the murder of a young female, a dancing girl. The evidence went to show that one of the accused was deeply in love with the girl, who, however, had rejected his addresses. On this the lover called in the assistance of the other accused persons, and asked them to prepare—at least, so it was urged for the defence—a love-philtre to be given to the girl, which would cause her to look favourably on his suit. The result was that a number of sweetmeat balls were prepared and distributed, with some ceremony, at an assembly where deceased and others were present. Apparently one only of these sweetmeat balls contained poison, viz. the one given to the deceased, as she and her brother, to whom she had given a portion of the ball handed to her, were the only persons who suffered from symptoms of irritant poisoning. After death twenty grains of arsenious oxide was found in the contents of deceased's stomach. On the whole, the evidence in this case was not inconsistent with the view, that the theory set up by the defence was true, and that there was no murderous intent.—(b) (*ib.* for 1878-79). The history of a case from Gadag, in which four persons—two children, who died, and two adults, who recovered—were poisoned by arsenious oxide, was thus given by the assistant-surgeon in charge of the dispensary :—"The mother of the deceased children obtained three small sweetmeat balls from one of her paramours, with the object of administering the same to a man named Dushrath Sonar, who was also in love with her. The man who prepared the balls, and gave them to the woman, states that he mixed some kind of white powder with the balls. The powder was procured by him from a mendicant Byragi, and the sweetmeat balls were made specially mixed with the powder to poison Dushrath Sonar; but the woman, instead of giving them to the Sonar to eat, kept them in her house. During the absence of the woman from her house on some business, her mother happened to find the sweetmeat balls, and, not knowing that they were poisoned, gave two of them to the children to eat; the third ball was eaten by herself and her other daughter, who was in the house."—(c) (*ib.* for 1876-77). The history accompanying a case from Amraoti was as follows :—"A man lost some ornaments, and suspected his sister of stealing them. Under the advice of a 'wise man,' he put outside his door a copper pot, with a lump of cowdung in it, advertising his friends that he had done so, and that if the thief put the ornaments into the pot, nothing further would be said. This failed. The 'wise man' thereupon assembled the neighbours, and an ordeal was instituted, each person being required to eat a small quantity of sugar." The result was that the sister died with symptoms of irritant poisoning, and one and a half grains of arsenious oxide was found in the contents of her stomach.

17. Apparently false confession of Arsenical Poisoning (Bo. Analyser's Rep. 1884).—In a case from Nasik a man died suddenly, and his brother accused deceased's wife of poisoning him. The wife was taken into custody, the body disinterred and examined; no signs of irritant poisoning were found. The viscera were forwarded for analysis. In the meantime the wife, while in custody of the police, confessed to having put arsenic into her husband's food, and some white substance, stated to have been produced by her, was, on examination, found to be arsenious oxide. On analysis, however, not the slightest trace of arsenic could be found in the viscera of the deceased.

18. Arsenical Poisoning. Homicide—quantity of poison found large (Browne and Stewart, 'Trials for Murder by Poison,' p. 358).—(a) The late Professor Christison, writing to the *Edinburgh Medical Journal*, December 1857, cited a case undoubtedly one of murder, where between 90 and 100 grains of arsenious oxide was found in the contents of the stomach of an adult. The poison was administered in whisky punch, with sugar, the arsenic being kept in suspension by constant stirring. Professor Christison's letter had reference to an argument put forward for the defence in the *Madeline Smith Case* (Edinburgh, 1857), viz., that as eighty-eight grains of arsenious oxide was found in the contents of the stomach of the deceased, the case must have been one of suicide. (b and c) Taylor (on Poisons, p. 157) mentions two other cases, both charges of homicide, in which, after death, the quantity of arsenious oxide found in the stomach was large, viz. *Reg.* v. *Dodds*, in which 150 grains was found, and *Reg.* v. *Hewitt*, in which 154 grains was found.

19. Arsenical Poisoning; accidental cases.—(a) In Bombay, some years ago, a number of school children were poisoned by swallowing portions of a mass of arsenious oxide which they had found lying on the ground.—(b) (Bo. Analyser's Rep., 1873-74). Five lumps of arsenious oxide, weighing together 128 grains, were found in the stomach of a woman who died in the Jamsetjee Jejeebhoy Hospital, Bombay. Her story was that she had eaten the arsenic in mistake for "Khadu" (pipe-clay). "Khadu" eating seems to be a common practice among Hindu females.—(c) (Taylor on Poisons, p. 354). In the Bradford lozenge case a confectioner, intending to adulterate lozenges with plaster of Paris, mixed with them a quantity of white arsenic, which had been supplied to him through mistake. More than 200 persons were poisoned, of whom seventeen died.

20. Arsenical Poisoning; external application.—(a) (Taylor on Poisons, p. 304). The mother of a girl, æt. nine, rubbed some white precipitate ointment, mixed with arsenic, on her child's head, in order to kill vermin. No symptoms appeared until the fifth day, when the child seemed unwell and complained of thirst. There was slight purging, with cramps on the eighth

day. Death took place on the tenth day. On post-mortem examination, inflammation of the mucous membrane of the stomach and duodenum was found.—(b) (Taylor's 'Manual,' p. 99; and Blyth on Poisons, p. 510). In 1876 a number of infants were poisoned in England by arsenious oxide contained as an adulterant in violet-powder. Some of the powder was found to contain over 38 per cent. of $As_2 O_3$. In one case reported by Dr. Tidy, the powder was applied to the skin of a newly-born infant, at intervals, for three days. The skin became intensely red, and ultimately in some parts assumed a sloughy appearance. The child died on the tenth day.—(c) (Taylor on Poisons, p. 11). Two shepherds were engaged in sheep-dipping for nine hours, using a mixture of arsenious oxide and solution of carbonate of potash. Both suffered; one, on the fourth day after the dipping, had eczema of the scrotum, and vesicles on the thighs, slight fever, and great thirst. Dr. Watson, who reported this case, states that other shepherds, even when using $As_2 O_3$ only, suffered from eruptions, chiefly on the hands, forearms, scrotum, and thighs.

21. The Baroda Case.—In this case "Colonel Phayre, the Resident at Baroda, experienced one morning a feeling of nausea, accompanied by salivation, and a peculiar taste, which he described as metallic; these symptoms came on about half an hour after he had swallowed a small quantity of sherbet, made of pumalo juice. Hastily concluding that the sherbet was bad, and had been the cause of the symptoms, he threw it away; but in the act of replacing the tumbler on the table, he noticed a sediment lying at the bottom of it. This sediment was proved, on analysis, to consist of arsenious oxide, mixed with finely-powdered diamond" (Dr. Wellington Gray, in Bo. Analyser's Rep. for 1874-75). It was alleged that this attempt to poison Colonel Phayre was instigated by the then Guicowar of Baroda, and a Court of Inquiry was held. One of the Residency Peons was suspected of having introduced the poison into Colonel Phayre's sherbet. On searching this man, a packet of arsenious oxide was found in his belt, and a sore was found on his skin, in such a situation as to make it possible that it had resulted from continued contact with the arsenic. Hence arose the question, Is the long-continued application of arsenious oxide to the skin liable to produce a sore thereon?

22. The De Ga Case (see Bo. Analyser's Report for 1872–73).—In this case a number of the members of a Portuguese family of position, resident in Bombay, were poisoned by arsenic. The circumstances of the case were as follows. A man, believed to be a Hindoo, who afterwards disappeared and has never been traced, left at the family residence in Bombay a present of cakes. Some of these cakes were passed on to another house, where other members of the family resided, and these, in their turn,

sent a portion to a third house. All who partook of the cakes suffered; three died. The cakes, on examination, were found to contain an interior layer of jam, mixed with a quantity of coarsely pounded arsenious oxide. In the course of the police inquiry into this case, suspicion arose that the poison had been abstracted from a particular druggist's shop in Bombay. It, however, turned out that the only arsenious oxide in this shop was a quantity contained in a jar. A portion of the contents of this jar, submitted to me for examination, proved to be entirely composed of minute unbroken crystals, much smaller in size than many of the fragments of arsenious oxide contained in the cakes. Hence it was highly unlikely that the poison found in the cakes had been obtained from the shop in question.

23. Cases of poisoning by the Sulphides of arsenic (Bombay Analyser's Reports).—(a) In Bombay several persons were poisoned, all of whom, however, recovered, by orpiment contained in food. The poison was introduced into the food in turmeric powder, which, on examination, was found to contain over ninety grains of orpiment per ounce.—(b) The wife of a man who was very ill with fever gave him, according to her own confession, orpiment, apparently in some *congy*. The husband died three days afterwards; traces of arsenic were found in his viscera. The post-mortem appearances were: much congestion and inflammation in patches, of the gastric mucous membrane; small intestines also inflamed, and in parts in a state approaching mortification; liver and spleen enlarged, and signs of commencing inflammation of both lungs.—(c) In another case orpiment was found in some cooked rice. A man who had partaken of a portion of this rice suffered from symptoms of irritant poisoning, but recovered.—(d) Several persons, all of whom recovered, were poisoned by bread made from flour in which, on analysis, orpiment was found.—(e) An apothecary, stationed in Sind, noticed some red powder at the bottom of a cup of tea he was drinking; this proved to be realgar. One of his servants, taxed with having attempted to poison his master, confessed to having put the powder into the tea at the instigation of a man, who told him that it was a charm which would lead to the early transfer of the apothecary.—(f) A woman confessed to having given to a child, who died from arsenical poisoning, some sugar mixed with rat poison. This rat poison proved to be a mixture of flour and realgar.—(g) The servant of a shopkeeper at Karachi brought to his shop, as part of a meal intended for him, an omelet, in which some red powder was visible; this turned out to be realgar.—(h) In a case in which two persons died from poisoning by arsenic, some flour and bread, of which they had eaten a portion, was found to contain mixed orpiment and realgar. —(i) In a case of attempted poisoning some sweetmeat, forwarded for examination, was found to contain both realgar and orpiment,

and, in another similar case, realgar and sulphate of copper were found in poisoned sweetmeat.

24. Cases of poisoning or attempted poisoning by the Arsenites of copper (Bombay Analyser's Reports).—(a) A woman was found in the act of putting something into a pot of drinking water standing in a Bhora's house. She was seized, and a packet found in her hand, in which was a quantity of aceto-arsenite of copper. In the struggle the pot got broken, and the water spilt, but aceto-arsenite of copper was found in some earth collected from the spot.—(b) A woman charged a man, described as her "kept husband," with an attempt to poison her, by giving her in lieu of "goolchund" (a sweetmeat containing Indian hemp and conserve of roses) a substance which, on examination, was found to consist chiefly of sugar and arsenite of copper.—(c) A child, about ten months old, was admitted into the J. J. Hospital, Bombay, suffering from symptoms of irritant poisoning, the result of sucking the paint off a toy parrot painted bright green with arsenite of copper.

25. Death from Arsenical poisoning. Complete elimination of arsenic from the body.—(a) (Taylor on Poisons, p. 335). A woman was charged with poisoning her husband by arsenic. The husband suffered from the usual symptoms of arsenical poisoning, and died from exhaustion on the fifteenth day. No arsenic could be found in the viscera of the deceased. Mr. Herapath, of Bristol, who made the analysis, stated that "neither in his reading nor experience had he known arsenic to have been detected so long as fifteen days after its administration."—(b) (Bombay Analyser's Report, 1874-75): "In a case from Ahmednagar, a man lived three days after a dose of about 75 grains of arsenic. He is said to have suffered during the whole time from purging and vomiting." Not a trace of the poison could be found in either the stomach or liver. Dr. Wellington Gray, who made the analysis, remarks in reference to this case: "It is quite possible that arsenic may have existed in the more distant tissues of the body, for the examination of which no opportunity was given." Arsenious oxide was detected in some bread, a portion of which had been eaten by the deceased.

26. Antimonial poisoning.—(a) **The Bravo case** (July, 1876). In this case Mr. Bravo, a young married man of good position, died undoubtedly from poisoning by tartar emetic. Deceased, on the evening he was attacked with symptoms of poisoning, stated—so one of the witnesses at the inquest deposed—that, owing to jealousy of his wife, he had taken poison. Shortly after this he became very ill, and medical assistance was sent for. Sir W. Gull, one of the physicians called in, stated that he told the patient that his symptoms were due to poisoning, and asked him how he came by it. He answered, "I took it myself." Asked what he had taken, he replied, "Laudanum." Told that

he must have taken more than laudanum, he said, "Before God, I only took laudanum." There was no evidence to show how deceased came by the tartar emetic, from the effects of which he died, and the coroner's jury returned a verdict of wilful murder against some person or persons unknown.—(b) **Reg. v. Smethurst.** (Browne and Stewart's 'Trials for Murder by Poisoning,' p. 448.) In July, 1859, Thomas Smethurst, a surgeon, was tried at the Central Criminal Court for the murder by poison of Isabella Bankes, a lady with whom he had contracted a bigamous marriage. Deceased, just before her death, made a will, leaving all her property to the prisoner. The medical witnesses for the prosecution deposed that, in their opinion, the cause of death was slow poisoning by some irritant, and on analysis traces of antimony were found in the viscera of deceased. For the defence, it was urged that several of the symptoms of slow poisoning by arsenic or antimony were absent; *e.g.*, there was no conjunctivitis, no skin disease, and no excoriations at the orifices. That the symptoms, &c., might have been due to acute dysentery. That the vomiting and diarrhœa from which deceased suffered might have resulted from her being in an early stage of pregnancy. That the traces of antimony found in the viscera (and a minute quantity of arsenic found in an evacuation passed by deceased) might have been due to the presence of arsenic in the bismuth, and of antimony in the grey powders, administered as medicines. The prisoner was convicted. Subsequently memorials, backed by the opinions of eminent medical men, were presented to Government in the prisoner's favour. The whole case was referred to Sir B. Brodie, and on his report the prisoner was pardoned.—(c) **Case of Dr. Pritchard** (*ib.* p. 397). In July 1865, Dr. Pritchard, of Glasgow, was tried for the murder of his wife and mother-in-law, the first by slow poisoning with antimony, and the second by poisoning with antimony and aconite. Antimony was found in the viscera of both. The prisoner was convicted, and subsequently confessed his guilt.

27. Mercurial poisoning. Fatal result attributed to the conversion of calomel into corrosive sublimate.—(a) ('Edinburgh Medical and Surgical Journal,' vol. liv., 1840, p. 250). A physician prescribed for a child powders, containing in each ½ a grain of calomel, 5 grains of ammonium chloride, and 5 grains of sugar. After taking a few of these powders the child died, with all the symptoms of poisoning by corrosive sublimate. The apothecary who made up the prescription was charged with causing the death of the child, it being supposed that he had by mistake substituted corrosive sublimate for calomel. This led to experiments being instituted, the result of which was to clearly show that calomel, by the action of ammonium chloride solution, is at the temperature of the body, and even at ordinary temperatures, decomposed, with formation of mercuric chloride and

metallic mercury. (b) **Bombay Chemical Analyser's Records.** In the year 1860 the following case was referred to Dr. Haines, then Chemical Analyser, Bombay, for opinion. A soldier in hospital at Canton, suffering from fever, was ordered pills, each containing 3 grains of calomel, two to be taken every two hours, with two spoonfuls of a mixture composed as follows:—Ammon. sesquicarb., ʒij; Liq. ammon. acet., ℥vj; Water, ℥vj. After two doses symptoms of gastric irritation came on, but two more doses appear to have been taken before the medicine was stopped. After several days' treatment the man died, and after death violent inflammation and ulceration were found in the stomach and pharynx. Experiments made by Dr. Haines with reference to this case showed that, at the temperature of the body, solution of acetate of ammonia decomposes calomel, with formation of mercuric chloride and metallic mercury, the action being more rapid if excess of carbonate of ammonia is also present.

28. Mercurial poisoning. Metallic mercury swallowed.—(a) (Taylor on Poisons, p. 360). For the purpose of causing abortion, a girl swallowed 4½ ounces by weight of mercury. It had no effect on the uterus, but in a few days she suffered from a trembling and shaking of the body (mercurial tremors) and loss of muscular power. These symptoms continued for two months, but there was no salivation, and no blue mark on the gums.—(b) (McLeod's Bengal Medico-Legal Report for 1869). The following case was referred to Dr. Bateson, Civil Surgeon, Umballah. An individual was charged with attempting to poison a woman by administering to her liquid mercury in food. The woman is said to have vomited twice after taking the mercurialised food, and to have had "a red swelling of the gums, with bleeding on pressure of the finger," attributed by the sub-assistant surgeon who saw the case to the action of mercury. At the first inquiry the charge broke down, owing to the sub-assistant surgeon stating that mercury (liquid metallic) was not a poison. A conviction, however, was subsequently obtained, apparently under sect. 328 of the Penal Code (see p. 111). Remarking on this case, Dr. Bateson says: "It seems it is customary, any way about here, to administer mercury in some thickish food to spite an enemy, to thwart an intrigue, or to punish accessories to one. The present case belonged to the last kind."—(c and d) (Bombay Chemical Analyser's Report, 1874-75). In two cases during this year metallic mercury was found in articles of food; in one 29 grains of mercury was extracted from a small quantity of sweetmeat, and in another 3 grains was extracted from some food.

29. Poisoning by Tylophora fasciculata (Bhui-doree).—Assistant-Surgeon Narayen Ananta, in charge of Pandharpur Dispensary, reported in April 1880 the following case:—A Mahomedan family, consisting of six adults and a servant boy æt. about four-

teen, were attacked soon after a meal with symptoms of poisoning. The servant boy died in about two hours. The others were seen the next morning, when they complained of dryness of the throat, great thirst, and a feeling of soreness over the whole body. Their pupils were dilated, and pulse full and slow. They stated that soon after taking their mid-day meal on the previous day they felt some tingling sensation in the mouth, followed by dryness of the tongue and throat and giddiness, and loss of power over the extremities. After this they became insensible. Three of them vomited and recovered consciousness at about 8 P.M.; the other three remained insensible till midnight. On post-mortem examination of the body of the boy, the following appearances were noted:—Face bloated, tongue and eyes slightly protruding, veins of the neck turgid. Lungs engorged; right side of the heart full, left empty. Slight congestion of the pia mater. A small patch of redness on the mucous membrane of the stomach. Accused in this case, it was stated, who was at enmity with the persons poisoned, asked a friend to recommend him something to kill rats with. The friend advised him to use bhui-doree. On this accused, it was reported, obtained some bhui-doree roots, and having reduced them to powder mixed this with some flour, from which subsequently the food eaten at the meal referred to was prepared.

30. Poisoning by Gloriosa superba.—Dr. Buttacharjee, 'Indian Medical Gazette,' 1872, p. 153, reports the following case:—A female, æt. eighteen, swallowed a quantity of the powdered root. Symptoms of poisoning appeared in half an hour, and were: retching, violent vomiting, spasms, and contortions of the body, with fearful racking pain; from time to time there were short intervals of relief, followed by recurrence of the same symptoms. Death took place in four hours. The post-mortem appearances were congestion of the brain and its membranes, with extravasations of blood. The lungs, liver, and kidneys were all deeply congested. The gastric mucous membrane showed signs of inflammation. The peritoneal covering of the fundus of the uterus (unimpregnated) was also found inflamed.

31. Poisoning by pounded glass.—(a) (Christison on Poisons, p. 654). Portal relates a case of a man who undertook for a wager to eat his wine-glass, and actually swallowed part of it. He was attacked with acute pain in the stomach, and subsequently with convulsions. The treatment consisted in the administration of bulky food (cabbage) followed by an emetic. The man recovered.—(b) (Chevers, p. 288, quoted from N. Sydenham Soc. Year Book for 1863, p. 401). A woman swallowed a quantity of coarsely powdered glass in order to "scour out" her stomach. Intense pain in the stomach came on, with tenderness; the pulse became small, 100 per minute; there was thirst and pallor. An emetic was given, and the next day the urgency

of the symptoms had passed off. The woman recovered.—(c) (Christison on Poisons, p. 653). In a case which occurred in Paris a woman, after a hasty dinner, became unwell, and next morning was seized with violent pain in the stomach and vomiting, and died in convulsions. Forty-two days after death her body was exhumed, black points and patches were found in the bowels, together with a quantity of broken glass. The medical witnesses differed as to the cause of death.—(d) (*ibid.* p. 654). In a case published by Mr. Hebb, of Worcester, a child, eleven months old, died of a few days' illness in very suspicious circumstances. On post-mortem examination the inside of the stomach was found lined with a tough layer of mucus streaked with blood. The villous coat was highly vascular, and covered with numberless particles of glass of various sizes, some of which simply touched, while others lacerated it. No other morbid appearance could be detected in the body.—(e) (Bo. Chemical Analyser's Report for 1875–76). A male adult was attacked with symptoms of irritant poisoning, and died in forty-eight hours. The mucous membrane of the stomach was found reddened, but not rugose. A quantity of powdered glass was found in the contents of the stomach. No irritant substance other than pounded glass could be found in the viscera.

32. Accidental poisoning by Carbon dioxide.—(a) (Ind. Med. Gazette for 1877, p. 184). Dr. Moffatt reports that four men were brought one morning to the dispensary at Naini tal in a state of insensibility. They had been found in a closed room 6 × 8 × 7 feet, with a pan of charcoal between them. The previous night had been a very cold one. Of the four one never recovered sensibility, and died shortly after admission. The other three are described as being, soon after admission, in a drowsy semi-conscious state; when shaken and spoken loudly to, they could be made to sit up and answer questions. Their eyes had a filmy look, the pupils were dilated, the pulse small and weak. Two of the three recovered completely; the third was attacked with œdematous erysipelas, and died four days after admission. (b) A very similar case, in which five persons were poisoned, of whom one died, occurred at Nowshera (see *ibid.* March, 1885).

33. Accidental poisoning by gases accumulated in a grain pit (Ind. Med. Gazette for 1874, p. 295).—Dr. Gardner, of Saharunpore, reports a case in which three men died shortly after descending into a pit used for the purpose of storing grain. The pit had just been opened, but instead of, as is customary, leaving it open for some time before allowing any one to descend, the owner being afraid of rain, sent his servants, four in number, down at once. The fourth man was also attacked, but recovered. The post-mortem appearances in the three fatal cases were those of death from apnœa, with numerous sub-pleural ecchymoses,

"averaging in size half a split-pea, some larger, some smaller, and of a dark purple colour, and about two lines or more in depth."

34. Severe symptoms arising from the introduction of opium into a wound (Chevers, p. 228).—A Burman boy was struck on the forehead, causing a gaping wound about an inch long. This the parents, about three hours after, stuffed with about forty-five grains of opium. On the third day he was brought to a medical man (Dr. H. W. Jones) in a semi-comatose state, but recovered under active treatment.

35. Hypodermic injection of Atropine in opium poisoning.—During the four and a half years ending November, 1885, sixty-four adults poisoned by opium were treated in the Jamsetjee Jejeebhoy Hospital, Bombay, by hypodermic injection of atropine in ¼ grain doses repeated at intervals, until dilatation of the pupils occurred. Of the 64, 31 died and 33 recovered. Of the 31 who died, 7 had been under 2; 11 over 2 and under 6; 8 over 6 and under 20; and 5 over 20 hours in hospital. The amount of atropine injected in the fatal cases was in 9 a ¼ of a grain; in 10 over ¼, but not more than ¾ of a grain; in 8 over ¾ and under 1½ grains; and in 4 over 1½ grains. The amount injected in the 33 cases of recovery was in 12 ¼ of a grain; in 12 more over ¼ to ¾ of a grain, and in 9 from over ¾ to 1½ grains.

36. Running Amok while under the influence of opium (Chevers, p. 789).—A poorbea, having a dispute with his nephew in regard to some property which terminated in litigation decided in the nephew's favour, armed himself with a straw chopper, and went in search of him. Not finding the nephew, he entered the nephew's house and severely wounded a woman, his nephew's wife, and her two children, killing one. Leaving the house, he then attacked and wounded a young man, who appears to have been simply a passer-by. He was shortly afterwards captured and put in confinement, but died during the day from the effects of the opium he had taken previous to committing the crime.

37. Homicidal violence committed while under the influence of Cannabis (Chevers, p. 790 *et seq.*)—(a) "One Madar Buksh, of Mirzapore, hacked his wife to pieces, inflicting twenty-two wounds with a sword, probably under the influence of jealousy. He confessed the crime, saying that he suspected the woman of infidelity, and that before his return home on the fatal night a man gave him a pill to eat; it was majoon, or bhang, and very powerful. The person named denied altogether that he had given the pill to the prisoner. The judge thought it not improbable that intoxication was resorted to, purposely to gain heart for the deed which was meditated."—(b) "One Kumla, a burkundauze of Boolundshur, killed, without any evidence of sufficient provocation, two boatmen who were ferrying him across the river. The

Sessions Judge believed that having been at a religious festival at Belown, the prisoner had probably indulged in bhang, or some intoxicating liquor, and being temporarily excited, was irritated by the slowness of the men. He was sentenced to death."—(c) Three Sepoys went over from Bhurtpore to witness the religious ceremonies at Goburdhun. Suddenly, and, as far as could be ascertained, without any provocation, one of them drew his sword, decapitated first one of his comrades, and then three women, entirely strangers to him. His defence was that he had taken bhang, and was beside himself. Before the magistrate he had said that the man whom he had killed had threatened to strike him. At the time he committed the act he apparently showed no signs of intoxication.

38. Running Amok.—(a) (Chevers, p. 792). A Mahomedan Khalasie, excited by jealousy of his wife, apparently determined to revenge himself on mankind in general. He seized a sword, rushed into the street, and attacked every one he met. Before he could be secured he had killed two children, and wounded, more or less seriously, two other children, and seven adults. —(b) (*ibid.* p. 791). A Havildar in the Kumaon battalion, while seated in the orderly room with other native officers of the regiment, suddenly rose and killed one Nardeb, an acting Havildar, with a kookree. He then rushed about and wounded two Sepoys of the corps. It was alleged that deceased had debauched the prisoner's wife.

39. Dhatura poisoning; road robbery.—(a) (Bo. Analyser's Report for 1878-79). Two merchants started with a tattoowalla, from Hubli in the Dharwar Collectorate, on an expedition to buy cotton in the neighbouring villages. As they were starting, a man and a woman offered themselves as guides, stating that they would show them where cotton was to be had. At a halt, food was prepared by the woman, of which the merchants and the tattoowalla partook; all three became insensible, and were robbed. One of the three died.—(b) (McLeod's Bengal Med.-legal Report for 1868-69, p. 113). Three adults and a lad were on a pilgrimage from Bombay to the North-west. At Hurdah they were overtaken by a man who said he was going in the same direction. He contrived to make himself very agreeable, and they all travelled together until they reached a village called Bansa, a few miles from Dumoh. Here they halted under the shade of a few trees beyond the village. According to the statement of the lad, the only survivor of the four, the stranger used daily, while on the march, to prepare sherbet for the party. He did so at this last halt, and after drinking the sherbet he had prepared, all four became insensible, and were robbed by the stranger, who disappeared, but was afterwards arrested in company with another party of travellers.—(c) (Taylor on Poisons, p. 776). Dr. Irving, of Allahabad, mentions a case of robbery by aid of

dhatura in which the poisoner, a man named Basower Singh, in order to lull suspicion, partook of the poisoned food himself, and died from its effect, while his victims whom he had robbed, recovered.

40. Dhatura poisoning; robbery.—(a) (Chevers, p. 155).—In 1852, two men, Joogul and Pertab, went at night to the house of a prostitute at Meerut, named Pearee. Joogul arranged to pass the night with Pearee, and, having paid her eight annas in advance, went away, but shortly afterwards returned with some sweetmeats, part of which he gave her. Pearee, soon after eating the sweetmeats, began to feel ill, and suspecting that she had been drugged, went to the door and informed a chowkeedar, who kept watch outside the house, and in the morning sent for the police. The police, on arrival, found Pearee insensible, and arrested Joogul in the house, and afterwards arrested Pertab. On the trial the prisoners confessed that they had arranged that Joogul should give the woman sweetmeats, with which dhatura had been mixed, so that they might rob the house while she was insensible.—(b) (Bombay Analyser's Report for 1883). The body of a man was found in a tank in Bombay. The feet were tied together, and the post-mortem appearances indicated death by drowning. Ornaments which deceased had been wearing a few hours before death were missing, and were subsequently traced to the possession of two men, in the company of whom deceased had been seen on the evening of his disappearance. On analysis, daturine was found in the contents of the stomach of the deceased, and one of the two accused is alleged to have stated that he had seen his companion put a powder into some liquor, which he gave to the deceased while in their company.—(c) (Bombay Analyser's Report for 1877–78). Four children were minding sheep in a field, when some persons offered them sweetmeats, which they ate; all became insensible, and the sheep disappeared. Two of the children died the same evening, and another on the third day; the fourth, who had vomited a good deal, recovered. Daturine was detected in the contents of the stomach in each of the three fatal cases.

41. Dhatura poisoning; motive other than theft.—(a) (Bombay Analyser's Report for 1884). In a case from Amraoti a man and his mother, after eating some food prepared by the wife of the former, were attacked by symptoms of dhatura poisoning; both recovered on the third day. The wife was taken into custody, and confessed to having put dhatura into some cooked bhaji, which she had given to her husband and his mother. A portion of the bhaji, sent for examination, was found to contain daturine. The accused also stated that she had put arsenic and aconite root into some bread, which she had given to the two sufferers. In neither case, however, were any symptoms of arsenic or aconite poisoning present, and no trace of either of

these poisons could be found in a portion of the bread submitted for examination.—(b) (Bombay Analyser's Report for 1884). —In a case from Bagevadi (Kaladgi District), daturine was found in some sweetmeat given by one man to another, who, after eating a portion, suffered from the usual symptoms of dhatura poisoning. The motive in this case was stated to be to make the victim "mad on account of kept women."—(c) In 1848, and again between 1866 and 1868, the Bombay Police used often to pick up in the streets of the city, on the same day, several persons suffering from dhatura poisoning. The affected individuals were mostly mendicants or others, apparently so poor as to make it unlikely that the motive of administration was facilitation of theft. Possibly, in these cases, the poison was given simply in order that the antics of the victims might afford amusement, or it may be that the motive for administration was connected with a belief, that the utterances of persons under the influence of dhatura are oracular, and may be depended upon as a guide to the success of undertakings, &c. As late as 1883, an apparently similar case was reported from Karachi, in which the victims were 18 in number, all Fakirs, who had been poisoned by dhatura, given to them in sweetmeat.

42 Accidental poisoning by daturine (Bombay Analyser's Report for 1876–77).—A child, two years old, got hold of a small pill-box, in which a small quantity of daturine was kept for use in ophthalmic practice by its father. Shortly afterwards the child became insensible and convulsed, the pupils became widely dilated, and death took place with well-marked symptoms of dhatura poisoning.

43. Sudden death in the first spasm of tetanus.—Chevers (Medical Jurisprudence, p. 248), under this heading, cites the following case :—" An apparently healthy boy, one of the pupils of the La Martinière School, Calcutta, was seated on his bed, having a small sore on his foot, dressed by the native doctor. Having applied the dressing, the native doctor was leaving. He walked straight to the door, but as he was passing out he heard a noise from the bed. Turning, he saw the boy supported on his occiput and heels, his body being arched up in opisthotonic spasm. He ran to the bed, the body sank, and death was immediate."

44. Cases of poisoning by aconite. Liquor drugged with aconite.—(a) (Chevers, p. 138). In 1834 about 70 men, 18 of whom died, were poisoned at Benares by drinking Mowra liquor obtained at a particular liquor shop. One of the servants at the liquor shop afterwards confessed to having put aconite root into the liquor.—(b) (Bombay Chemical Analyser's Report for 1884). In a case from Morsi (Hyderabad Assigned Districts) a small bundle, taken out of the receiver of a country liquor still, was on examination found to contain a quantity of fragments of aconite root. Of eleven persons, who had drunk

liquor, bought at the shop of the owner of the still, ten, it was stated, suffered from slight symptoms of aconite poisoning; the eleventh, who had drunk about a quart of the liquor, died.—**Accidental cases**: (c) (Bombay Chemical Analyser's Report for 1875–76). In a case from Coompta a woman, after taking medicine, given to her by a quack for menorrhagia, suffered from "restlessness, depressed and irregular heart's action, coldness of the surface, numbness and tingling of the lips, tongue, and extremities, spasm of the diaphragm, and difficult respiration." The woman recovered under treatment; aconitia was detected in the vomit.—(d) (*ibid.* for 1877–78). A man was admitted into the Jamsetjee Jejeebhoy Hospital, Bombay, suffering from symptoms of aconite poisoning. On inquiry, it turned out that he had been taking pills, supplied to him by a native hakim. Some of these pills, on examination, were found to contain cinnabar and aconite.—(e) (*ibid.* for 1879-80). At Mahad, in the Colaba district, a man, the servant of a native Hakim, swallowed some of a preparation of aconite root he was making for his master; death resulted, and on analysis, aconitia was found in deceased's viscera.—**Homicidal cases**: (f) (Chevers, p. 140). "In 1854 one Anunchunder Roy, having incurred the censure of his family by his dissipated and extravagant mode of life, conceived the idea of murdering them all. For this purpose he purchased about an ounce of aconite root. He was seen pounding some of the root on a brick, and was proved to have deposited the powder in a utensil, containing a vegetable broth, at his brother's house. The brother and three women partook of the broth." The brother "was taken ill almost immediately, complaining of a burning sensation in his throat and stomach, vomited once, and expired during the night." The three women suffered from similar symptoms, but recovered.—(g) (Harvey's Bengal Report, p. 277). In a case reported from Gujranwala by Mr. R. C. Bose, a woman confessed to having killed her son-in-law, æt. 30, by administering to him half a tola of aconite root. "She said that, about half an hour after the imbibition of the poison, the man began to complain of pain in the stomach, numbness, tickling sensation in the throat, and subsequently of severe vomiting, followed by coldness of the extremities, collapse and rigidity of the hands and fingers. Death took place in seven hours."

45. Accidental poisoning by aconite. A typical case. (Chevers, p. 144.)—A Mahomedan, aged 35, was admitted into hospital, having about half an hour previously eaten by mistake a small portion of aconite root. "Immediately after chewing it, he felt a sweetish taste, which was soon followed by tingling of the lips and tongue, numbness of the face, and severe vomiting." On admission "he was extremely restless, tossing his limbs about in all directions, and constantly changing his position.

He complained of a burning sensation in the stomach, and a tingling and numbness all over the body excepting the legs. The tingling was specially marked in the face and tongue, so much so that he was constantly moving the latter to and fro, in order to scratch it against the teeth. Retching and vomiting occurred almost incessantly, and he constantly placed his hand over the cardiac region. His face was anxious, the eyes suffused, the lips pale and exsanguine, the eyelids swollen, pupils moderately dilated and insensible to the stimulus of light. Respiration laboured, 64 a minute; pulse, small and feeble, 66." There was inability to walk, but the man was perfectly conscious. 3¾ hours after admission, the symptoms had increased in severity, the pulse was intermittent, the tongue red and swollen, the tingling and numbness had extended to the legs. The points of a pair of scissors, four inches apart, applied to the skin of the arms and forearms, were felt as one. Applied to the thighs and legs, the two points were felt distinctly until the distance between them was reduced to less than 2¾ inches. About 9 hours after admission he began to improve, gradually recovered, and was discharged well in a few days.

46. Poisoning by aconite. Treatment by digitalis. Recovery. (Apothecary, Jas. Forsyth, Kasauli; Indian Medical Gazette, December, 1882.)—In this case the patient, who had been given aconite root by a Hakim as a cure for fever, was brought to hospital in an almost moribund condition, but recovered under the following treatment:—On admission 20 minims of tincture of digitalis was subcutaneously injected, and 15 minims more a quarter of an hour afterwards. A draught, containing brandy, carbonate of ammonia, and 1 drachm of tincture of digitalis, was given about one hour after the injection, and another drachm of tincture of digitalis in three doses during the next 2½ hours.

47. Poisoning by Nerium odorum. (Mr. Broughton, Civil Surgeon, Kolapore, quoted by Chevers, p. 256.) A man, æt. 35, after a quarrel with his wife, attempted suicide by swallowing rather more than an ounce of expressed oleander juice. After swallowing the poison, he is stated to have almost immediately fallen down insensible, and when admitted was insensible with flushed face and stertorous breathing. There were violent spasmodic contractions of the muscles of the entire body, more developed in the upper than in the lower extremities, and on the left more than the right side. "During the intervals of spasm the patient lay evenly on his back; and when action commenced, the superior contraction of the left side threw him over on his right." After some hours the spasms decreased, the pulse sank to a thread, and the extremities became cold. Insensibility lasted about 36 hours. Under active treatment, however, the patient ultimately recovered.

48. Poisoning by Nerium odorum. (Dr. Murray, Indian Medical Gazette, 1877, p. 319.)—Male, æt. 35. Quantity taken: a strained watery decoction of 4 ounces of the root. Death on the fifth day. Was attacked soon after swallowing the poison with vomiting and cramps; insensibility came on in two or three hours. Eight hours after swallowing the poison is described as insensible, skin cold and clammy, pulse weak and thready; muscles of the jaws stiff; eyes turned up, whites only visible; hands pretty open, but fingers rigid, thumbs turned inwards. During the night had frequent convulsive spasms, and had not recovered sensibility when taken away from hospital by his friends 48 hours after swallowing the poison.

49. Homicide by snake bite. (Fayrer's 'Thanatophidia of India,' p. 51.)—In this case, two snake charmers were convicted at Hurdah, in 1869, of causing the death by snake bite of three men. The prisoners, it was proved, partly by threats, and partly by assurances that it was in their power to prevent ill effects following, induced four men to allow themselves to be bitten by a Krait. All four were poisoned, one only recovering. What the motive for the crime was did not appear.

ILLUSTRATIVE CASES—PART C.

(See Chapters XXVI. to XXXII.)

Obstetric Jurisprudence and Rape.

1. A nullity of marriage case. (Tidy's 'Legal Medicine,' ii. p. 102.)—In the case of L. *v.* L., it appeared that the woman was impotent, but that she might possibly be cured by an operation involving no great risk of life, to which, however, she refused to submit. The Court, in granting the decree, said that it could not compel her to submit, and the man can only be expected to take all reasonable means to persuade her. This he has done, and she has distinctly refused (L. R. 7, P. Div. 16).

2. Procreation by cryptorchid. (Taylor's Princip. ii. p. 288.)—A man, in whom the testicles had not descended at the age of 30, had been twice married, and had had children by each wife, besides illegitimate children which were affiliated on him during the time he lived in service.

3. A similar case (*ibid.*).—Case of a man in whom the testicles had not descended, reported by Mr. Poland. This man married when he was 20, had two children by his first wife; and at the time of his admission into hospital (for hernia), had been married two years to a second wife.

4. Procreation by a monorchid. (Case of John Bury, temp. Queen Elizabeth, Guy, p. 40.)—"Willimet, the first wife of one John Bury, alleged that he was impotent; and on inspection by two physicians, he was found to have but one testicle, the size of a small bean, while she was a virgin. On this and other circumstantial evidence, the Ecclesiastical Court annulled the marriage. But Bury took a second wife, by whom he had a son."

5. Procreation after an attack of hemiplegia. (Guy's For. Medicine, p. 42.)—"E. K., æt. 58, when 33 years of age, had a well-marked attack of hemiplegia of the right side, which has left him lame and with his speech slightly affected. He alleges that he had connection with his wife within a week of his seizure, that his sexual powers have not been impaired, and that since his attack he has had three children, always considered as his own. His wife gives three weeks as the extreme limit of time after the attack, at which connection took place."

6. Alleged impotence in respect to one female, but not to others. (Woodman and Tidy, p. 679, quoted from Hargraves'

'State Trials,' i. p. 315.)—"In the reign of King James I. of England, the Earl of Essex was sued by his Countess for divorce on the ground that he was impotent. She claimed to be a *virgo intacta*, but is said to have substituted one of her maids for herself when examined. The Earl appears to have admitted the charge as regards the Countess, although he denied it *quoad* others."

7. A similar case. (Ogston's 'Lectures,' p. 81, quoted on authority of Pitaval.)—"Two gentlemen of rank, very much of the same age and personal appearance, were married to wives who proved unfruitful after several years of marriage. The two couples at last determined to proceed to a celebrated watering-place, in the hope of deriving some benefit from the change and the use of the springs. On the way they put up at an inn and retired for the night. But the two wives had preceded their husbands to bed, and each of the latter mistook his friend's room for his own. In consequence of the mistake each of the ladies proved with child."

8. Hysteria a cause of impotence in the female. (Tidy's 'Legal Medicine,' ii. p. 102, *G.* v. *G.*, L. R. 2 P. & D. p. 287.)—"In this case, where the parties had cohabited for two years and ten months, and the man's capacity and desire to consummate were not questioned, the Court being satisfied of the *bona-fides* of the suit, and of the practical impossibility of consummation in consequence of the hysteria of the woman, pronounced a decree of nullity, although there was no structural defect in the woman."

9. A similar case. (Tidy's 'Legal Medicine,' ii. p. 103. Case of *H.* v. *P.*, L. R. 3 P. & D. p. 126.)—"In a suit for nullity of marriage, it appeared from the husband's evidence that whenever he attempted to have intercourse with his wife the act had produced hysteria on her part; and that, although he had cohabited with her for more than three years, the marriage had never been consummated. The wife refused to submit to inspection. Decree *nisi* granted."

10. Two women afterwards found to be prostitutes; result of examination as to virginity doubtful in the case of one. (Guy's Forensic Medicine, p. 49, quoted from Parent-Duchatelet.)—Two young women, of genteel appearance, were attacked in the public streets by some young men, who called them opprobrious names, and told the passers-by that they were no better than common prostitutes. Some good-natured persons resented this conduct and took the girls' part, and a complaint was lodged on their behalf against their defamers, who were summoned before a magistrate. The defendants pleaded a justification, while the females, on the contrary, stoutly insisted on their purity, and even offered to submit to inspection by a medical examiner, which the opposite party dared them to do. A sworn inspector,

clever and conscientious, was appointed by the magistrate, and reported that it was totally out of his power to say anything certain as to one of the females; she might or might not be a virgin; but that the other had probably had some intercourse with men, though he could not assert the fact positively. Yet it subsequently came out that these young women had actually been for some time on the registers of the Police, and had both had repeated attacks of the venereal disease.

11. Evidence of virginity brought forward in disproof of alleged adultery. (Taylor, ii. p. 441: *Frazer* v. *Bayley*, Common Pleas, February 1884.)—It was alleged by defendant that the plaintiff, a married man, had had adulterous intercourse with a young woman, and that at an antecedent period she had left her home for the purpose of giving birth to a child privately. The late Dr. Ashwell was called upon to examine the woman, and he deposed that, in his opinion, she was a virgin and had never had a child.

12. A similar case. (*Ibid.*, *Delafosse* v. *Fortescue*, Exeter Lent Assizes, 1853.)—In this case, which involved an action for defamation of character, the plaintiff, a married man, æt. 64, had been charged with committing adultery with a certain woman. Several witnesses for the defendant positively swore that they had seen these persons in carnal intercourse. This was denied by the plaintiff; and as an answer to the case, medical evidence was tendered to the effect that the woman, with whom the adulterous intercourse was alleged to have taken place, had been examined, and the hymen was found intact. In cross-examination, however, this was admitted not to be a conclusive criterion of virginity, and a verdict was returned for the defendant.

13. Vulval penetration held to be sufficient to constitute rape, according to Indian law. (*Reg.* v. *Ferrol*, Bombay High Court Sessions, February 1879.)—In this case the prisoner was charged with rape on a child, six years old. The child had not complained, and admitted on cross-examination that she had not been hurt. The medical evidence proved there was no injury to the parts. The child was found to be suffering from gonorrhœa, so was the prisoner. It was clear that the penetration (if any) had been only vulval. Green, J., directed the jury that this was sufficient to constitute rape, and the prisoner was convicted of rape.

14. Conviction for rape in a case where consent of the female to sexual intercourse was given under misconception of fact. (From Tidy's 'Legal Medicine,' ii. p. 244; *R.* v. *Flattery*, L. R. 2 Q. B. D. 410.)—"The prisoner professed to give medical and surgical advice for money. The prosecutrix, a girl of nineteen, consulted him with respect to an illness from which she was suffering. He advised that a surgical operation should be performed, and under the pretence of performing it had carnal

connection with the prosecutrix. She submitted to what was done, not with any intention that he would have carnal connection with her, but under the belief that he was merely treating her medically, and performing a surgical operation, that belief being wilfully and fraudulently induced by the prisoner. The Court for Crown Cases Reserved were unanimously of opinion that these facts constituted the crime of rape."

15. Rape by one man unaided on an adult female. (From Casper's Handbook, vol. iii. p. 311.)—"L. persuaded F., a girl aged twenty-five, to accompany him to the Thiergarten in the dark, and after he had been baulked by her struggles in his endeavours to violate her against a tree, he seized her round the body and flung her on the ground, and being now, as she states, deprived of the power of resistance, he flung her dress over her head and violated her. Nine days subsequently I had to examine her. Her appearance was timid and maidenish, and without dissimulation she was deeply moved by what had befallen her. The entrance to the vagina was still reddened, and painful when touched and dilated; the hymen was completely torn, and bright red; carunculæ, still slightly swollen, were visible; the frænulum still existed. Without any leading question, and only in answer to general queries as to her bodily and mental condition, she declared that still a little, and several days ago much more, she could only with difficulty walk and pass urine and fæces. After carefully considering all that required to be considered in such a case, I came to the conclusion that a rape had actually been committed upon F. At the time of the trial, circumstances came out which only served to confirm this opinion. The police-officers who had hurried up at the cries of F. testified that the ground upon which she had been thrown was hard frozen, and they deposed that L, when arrested, and after his lust had been satisfied, was still in a condition of actual satyriasis."

16. Rape by one man unaided on a married girl æt. sixteen. (From Chevers, page 702.)—Fadil Gazee, of Jessore, a tall and powerful man, seeing a married girl of sixteen standing at her door, accosted her, apparently under the pretence of asking for a *soot nullee,* and wished to worm out of her if she was alone; finding that she was, he put his arms round her, forcibly drew her into the house, flung her down, and, gagging her mouth with her right hand, effected a criminal connection with her. Before her mouth was well gagged she managed to yell out for assistance, and her cries brought her mother and a neighbour, who found him in the act. He then got up hastily, offered to give her a rupee if she would say nothing more of the matter, and ran off.

17. Alleged violation during mesmeric trance. (From Taylor, ii. p. 447.)—"A girl (æt. eighteen) consulted a therapeutic magnetiser as to her health. She visited him daily for

some days. Four and a half months afterwards she discovered that she was pregnant, and made a complaint to the authorities against the magnetiser. They directed a physician and surgeon to determine the date of her pregnancy, and whether complainant might have then been violated and rendered pregnant contrary to her will, *i.e.*, if her volition could have been completely or partially annihilated by magnetism. The medical inspectors were satisfied that the pregnancy did not extend farther back than four and a half months; and founding their opinion on M. Husson's report, made to the Academy in 1831, concluded that, as a person in magnetic sleep is insensible to every kind of torture, sexual intercourse might then take place with a young woman without the participation of her will, and without her being conscious of the act, and consequently without her being able to resist the act consummated on her. This opinion was confirmed by that of Dévergie ('Gazette Médicale de Paris,' and 'Edin. Month. Jour.,' December 1860, page 566). There is another view of this case which does not seem to have occurred to the French medical experts, namely, '*Non omnes dormiunt quæ clausos habent oculos.*'"

18. Alleged violation during profound natural sleep. (From Ogston, page 121.)—A servant woman at an hotel in Nenagh proved pregnant, and solemnly declared that she was not conscious of having had intercourse with any man. Suspicion, however, fell upon an ostler in the establishment, who subsequently acknowledged that he believed he was the father of the child; that, having found the woman in a deep sleep from fatigue, caused by long-continued exertion and being kept out of bed two or three nights in succession, he had connection with her, and, as he believed, totally without her knowledge, as she did not evince the slightest consciousness of the act at the time, or recollect of its occurrence afterwards. The parties were married with mutual consent.

19. Another case. (From Taylor, ii. p. 445.)—Casper met with a solitary case in which a girl, æt. sixteen, accused a man of having had intercourse with her while she was sleeping in her bed, of which she was not conscious until he was in the act of withdrawing from her. On her own statement she was *virgo intacta* up to the date of this occurrence. Upon the facts of the case Casper came to the conclusion that, if her statement was true, the man could not have had intercourse with her without causing pain and rousing her to a consciousness of her position. The hymen was not destroyed, but presented lacerations in two places. This and other facts showed that there had been intercourse, but did not prove that this had taken place without the consciousness of the woman.

20. Death following rape. (Harvey, Ind. Med. Gaz., Nov. 1875.)—Rape on a female æt. nine. Death from hæmorrhage

from a wound on the genitals ascribed to the introduction of the male organ.. The left wall of the vagina was ruptured from the orifice upwards for 2½ inches, and the rent was an inch wide.

21. Injury to the genitals of a young girl for purpose of supporting a false accusation. (Gribble, page 173, quoted from the 'Medical Times and Gazette,' May 21, 1859.)—A procuress brought a girl into the officers' barrack, Fort William, Calcutta, but the person to whom she was presented objected to the girl on account of her youth. The bawd, having been disappointed of her fee, injured the child so as to cause very considerable hæmorrhage from the genital organs. The girl was seen by an assistant-surgeon in the Fort, and the circumstances of the case having been reported to the police, it was discovered to be a conspiracy against the officer to obtain money. The child recovered.

22. Post-mortem appearances of noma pudendi in a girl æt. five. (From Taylor, ii. p. 432.)—"The genital organs externally, and the skin around and beyond the anus, were intensely inflamed, swollen and ulcerated, and in an approaching state of gangrene or sloughing. The hymen was destroyed posteriorly, and the lining membrane of the vagina and uterus was much inflamed, of a dark purple colour, with softening and disorganisation of substance. The upper inguinal glands were enlarged on both sides. The child was in a neglected and dirty state."

23. Pregnancy a motive for murder or suicide. (From Bombay Chemical Analyser's Report for 1884, reported by the District Magistrate of Bassim, Hyderabad Assigned Districts.)—A widow seven months gone with child died rather suddenly; an inquest was held by the police, and a verdict returned of death from dysentery. Suspicion, however, being excited, a post-mortem examination was ordered, the result of which was the discovery of the pregnant condition of the woman (which had been concealed in the inquest report furnished by the police), and of the fact that the cause of death was arsenical poisoning. The District Magistrate remarks, in reference to this case, that there is every reason to believe that all engaged in the inquest tried to conceal the true cause of death.

24. Another case. (*Ibid.*)—In this case, which occurred in the Surat District, as in Case 23, the cause of death was arsenical poisoning, and the deceased was a widow far gone in pregnancy. The brother and sister of the deceased confessed to having given her eight annas' worth of opium in order to procure abortion or cause death, so as to avoid the disgrace arising out of her condition. No opium, however, could be discovered in the viscera of the deceased.

25. Pregnancy a motive for murder. (Chevers, p. 733.)—Allabux, of Purneah, was convicted of murdering his brother's

widow. He confessed that, having administered drugs to her in order to cause her to abort, and having failed, he and others took her to a river bank, put a cloth into her mouth, held her down and murdered her by cutting her throat.

26. Another case. (Bombay Chemical Analyser's Report, 1876–77, p. 18.)—Case of poisoning by arsenic reported by medical officer, Tatta Sind. "Deceased was promised in marriage to a man of her caste (viz. Mussulman), but before marriage she cohabited with him and became pregnant, and was advanced to above the fourth or fifth month, when her parents, to avoid disgrace, it is said, tried very much to procure abortion, but failed (much against her intended husband's will); so having failed to procure abortion, her parents, to save their reputation, it is suspected, gave her poison in her food."

27. A viable child born one hundred and seventy-four days after marriage. (The Jardine Case: Guy, page 131.)—The Rev. Mr. Jardine was married on the 3rd of March, 1835, and on the 24th of August following his wife was delivered of a girl, who, supposing her to have been the fruit of sexual intercourse on the day of the marriage, was only one hundred and seventy-four days, or five calendar months and twenty-one days old. The infant, which was undoubtedly immature, though to what degree could not be determined, died on the 20th of March, 1836, having survived about seven months. On this Mr. Jardine's parishioners brought a charge of incontinency against him before the General Assembly of the Church of Scotland, alleging that he must have had intercourse with his wife before marriage. The main question in the case was, Was it possible that a child not more than one hundred and seventy-four days old at birth, could be maintained alive for seven months? The Court found the charge "not proven."

28. Proof of live birth in a tenancy by courtesy case. (*Fish* v. *Palmer*: Taylor, ii. p. 207.)—The wife of the plaintiff, who was possessed of an estate in her own right, died after having given birth to a child. The child was supposed to have been born dead, and the estate was surrendered to the defendant, her heir. Ten years afterwards facts came to the knowledge of the plaintiff which led him to believe that the child was born alive, and that he had therefore wrongfully surrendered the estate. The evidence of live birth was as follows: it was proved that the accoucheur in attendance (who had died before the trial) had, an hour before the child was born, declared it to be alive, and ordered a warm bath to be prepared for it. Further, two women, who after the child was born placed it in the bath, swore that they twice saw a twitching or tremulous motion of the lips of the child. This motion of the lips was the only sign of life observed, but it was held sufficient to establish the fact of live birth.

29. Tenancy by courtesy in India. (*Sarkies* v. *Prosonomoyee Dosee*, I. L. R. 6 Calc. p. 794.)—In this case the widow of an Armenian, married before the Dower Act 29 of 1839, was held to be entitled to dower out of her husband's lands. In the course of the argument, Pontifex, J., remarked: "It would take away from the mutuality of contract between husband and wife to hold that the widow is not entitled to dower as against a purchaser from her husband. The husband is entitled to an estate by the courtesy in his wife's lands."

30. Two closely following acts of intercourse in the same female; both prove fruitful. (Guy's For. Med., p. 138, one of several similar cases quoted by Beck.)—"A female at Charleston, in South Carolina, was delivered in 1714 of twins within a very short time of each other. One was black and the other white. She confessed that on a particular day, immediately after her husband had left his bed, a negro entered her room, and by threatening to murder her had connection with her."

31. Supposed superfœtation. (*Ibid.*, p. 139.)—The wife of Raymond Villard, of Lyons, eight months after a previous abortion at the seventh month, was delivered of a living female child. "This delivery was not followed by the usual symptoms, no milk appeared, the lochia were wanting, and the abdomen did not diminish in size. Three weeks after her delivery she again felt the motions of a fœtus, the abdomen increased in size, and five months and sixteen days after delivery she was again delivered of a living daughter." Both children were alive two years after the birth of the first child. "Dr. Desgranges, who attended the case, adds to his report that the second child could not have been conceived after delivery of the first, inasmuch as no sexual intercourse took place between the husband and wife until twenty days after the first delivery," or four months and twenty-seven days before the birth of the second child.

32. Superfœtation? (From Tidy, ii. p. 149, quoted from Naphey, 'Physical Life of Women,' p. 156.)—"Marie Anne Bigaud, æt. thirty-seven, gave birth on April 30, 1748, to a full term mature boy, which survived its birth two and a half months, and to a second mature child (girl) on September 16, 1748, which lived one year. The interval between the two births was thus four and a half months (= one hundred and thirty-nine days). The mother, after her death, was proved not to have had a double uterus."

33. Viability of a one hundred and seventy-five days' child. (Dr. Outrepont's case, from Guy, p. 136.)—In this case "the mother, a young woman who had always been perfectly regular, menstruated as usual ten days after her marriage, and was subsequently repeatedly connected with her husband. About a fortnight after this menstruation she became changed in

appearance, and for the first time in her life had frequent attacks of vomiting and fainting. These symptoms continued, but the catamenia did not return, and about twenty weeks after their last appearance she felt the first movements of the child. Five weeks after this (*i.e.*, twenty-five weeks or one hundred and seventy-five days from the last appearance of the catamenia), she was seized with labour pains and hæmorrhage, and Dr. Outrepont having ascertained that this proceeded from the placenta being attached to the os uteri, encouraged the labour and brought it to a prosperous conclusion." The appearance of the child, a boy, at birth corresponded to those of a fœtus not more than twenty-five weeks old. He was known to be alive eleven years after the date of his birth.

34. Identity of human remains; ? signs of previous delivery present. (*R.* v. *Wainwright:* Taylor's 'Manual,' p. 495.)—The prisoner was tried for the murder of a woman with whom he had cohabited, and who had two children by him, the last being born about nine months previous to the time of her supposed murder. A year after her disappearance the mutilated remains of a female were discovered buried in premises belonging to the prisoner. Examination of these showed the uterus to be enlarged and flaccid; its walls were unusually thin. There were one or two white lines in the skin of the lower part of the abdomen, and other marks of a darker colour in the inguinal region. Two medical men who had examined the remains, were of opinion that they were those of a woman who had borne a child. Dr. Alfred Meadows, called for the defence, was of the contrary opinion, but stated that he believed it to be impossible to decide this question in any case with certainty.

35. Vagitus uterinus. (Ogston's Lectures, p. 247)—"In 1834 Dr. Jobert was called to assist in the delivery of a woman with a deformed pelvis, who had had two abortions previously. After strong pains the membranes had ruptured forty-eight hours before his visit. On examining the woman he found the head of the child above the brim of the pelvis, the occiput and face towards the right and left iliac fossæ. The parietal bones had alone entered the brim of the pelvis. The os uteri was dilated to about 2 inches. As the narrowness of the antero-posterior diameter of the pelvis proved an obstacle to the descent of the head, the forceps was applied to it, when, at the moment the operator commenced his attempts at extraction, the fœtus, during some seconds, uttered repeated and distinct cries which were heard by all in the room. After this, while considering whether it would be advisable to bring down the feet, from the little effect produced by the forceps, the cries were renewed as distinctly as before, as from the effect of repeated inspirations. Finally, when introducing his hand in order to lay hold of the feet, the moment it passed over the left shoulder, the fœtus for

the third time uttered cries less prolonged than before, yet sufficiently loud to be heard by all present."

36. Fractures of the skull of a fœtus in utero caused by a fall. (Quoted by Casper, iii. p. 115, on the authority of Masckka.)—A woman towards the end of the eighth month of her pregnancy leaped down from the second story, broke both her thigh bones, and died in six hours. On the fœtus in utero there were several fractures of both parietal bones, with extravasation of blood and coagula both on the external surface and within the cranial cavity.

37. Sudden delivery in a primipara. (Taylor, ii. p. 399, quoted from the *Lancet*, i. 1854, p. 637.)—M. C., æt. twenty-three, single, was suddenly delivered of a full-grown male child at 5.30 A.M. She stated that between 4 and 5 A.M. she felt griping pains. She suspected that her labour was coming on, and walked to a friend's house 600 yards distant to be confined. When she had proceeded half-way, she was suddenly delivered while in the erect position, and her child fell upon the pavement. The navel-string was ruptured transversely 4 inches from the navel, and the placenta was expelled. She walked to the place where she intended to be confined carrying the child, which she had wrapped in a petticoat. *This was her first child;* it was well nourished and healthy-looking. The only injury it had sustained by the fall was on the left parietal bone at the junction with the coronal suture; there was here a soft tumour between 2 and 3 inches in its transverse diameter, which was slightly ecchymosed. Both mother and child did well, and the tumour entirely disappeared at the end of three weeks. The cord was tied after the woman's arrival at the house.

38. Fracture of an infant's skull from attempts to aid delivery. (Taylor, ii. p. 404.)—Dr. Hicks was called by a midwife to aid the delivery of a woman. On examination he observed that the skull was fractured through the parietal bone of one side, and there was a slight fracture of the edge of the occipital bone, with a scalp tumour. The head of the child was at the brim of the pelvis, and the fractures had been produced by the midwife in her attempts to push the head back into the cavity.

39. Unconscious delivery during sleep in a primipara. (Chevers, p. 767.)—Dr. W. Case, of Chicago, attended a primipara whose delivery took place during profound sleep. During the day on which delivery took place she had been feeling unwell, but attributed this to over-fatigue on the previous day. Delivery took place rapidly, and the woman after it was complete, woke up in a fright, having dreamt that something was the matter with her.

40. Exertion after delivery. (Chevers, page 745.)—A Brahminee widow, after walking two miles, was delivered of a

mature male child, and leaving it in the dry bed of a stream walked back to the house from which she had started. She was tried for exposing her infant, and the Judge held that the facts of the case were inconsistent with her defence, viz., that she was in bad health at the time, and from bewilderment and pain was unaware of what she was doing.

41. Alleged abandonment of an infant. (*R.* v. *Beejoo*, 1st Mad. Sess. 1869, Mayne's 'Penal Code,' p. 275.)—In this case the following facts arose: A. the mother of a newly-born child, being herself too ill to move, sent B. to expose it. It was held by Scotland, C. J., that A. could not be convicted under this section, as she had not actually exposed the child, nor B. as she was not the mother. Also, that neither A. nor B. could be indicted for abetting the other, since as neither could have committed the offence there could be no abetment by the other. Of course, a person who has the custody of a child merely for the purpose of exposing it, cannot be indicted as a person "having the care of such child."

42. Failure of external violence to cause miscarriage. (Woodman and Tidy, p. 754, quoted from Tardieu.)—In the Assize Court of the Loire-Inférieure it was proved that a peasant who had seduced his servant and wished to make her abort, mounted on a strong horse, and put the girl on the same horse, then galloped wildly hither and thither, throwing her down on the ground whilst in full gallop, and this repeatedly. Having tried this twice without success, he applied to her stomach bread just taken from a very hot oven. This means failed like the former, and the poor victim gave birth to a living and well-formed child at term.

43. Failure of violence to cause miscarriage. (Guy, p. 85, on the authority of Dr. Wagner, of Berlin.)—A young woman seven months with child had employed savin and other drugs to produce miscarriage. As these failed, her paramour bound a strong leather strap tightly round her body. This, too, availing nothing, he (by his own confession) knelt upon her with all his weight, and trampled on her while she lay on her back. As this also failed, he took a sharp-pointed pair of scissors and proceeded to perforate the uterus through the vagina. Much pain and hæmorrhage ensued, but did not last long. The woman's health did not suffer in the least, and pretty much about the regular time a living child was brought into the world, without any marks of external injury upon it.

44. Miscarriage caused by drugs combined with mechanical violence. (Bombay Chem. Analyser's Report for 1884.)—In a case from Dohad (Panch Mahals) a quantity of orpiment and ruskapoora—impure calomel—were discovered among matters found in the house of some persons charged with procuring abortion. In this case a woman was heard screaming in a house;

the police entered, and found that the screams proceeded from a woman who had aborted, and who on examination told the following story. She said she had been forced against her will to take some medicine brought by the man in whose house she was found. After taking it she suffered from vomiting, and, in about two hours, from pain in the abdomen. As soon as the pain became severe, the man who gave her the medicine seized hold of her hands and feet, and his wife pressed on her abdomen, with the result that abortion was effected.

45. Post-mortem delivery. (Dr. Wright, Civil Surgeon, Jaunpur, Ind. Med. Gaz. 1887, p. 296.)—A Mussulmani, aged about twenty-seven, at about the full term of pregnancy, committed suicide by drowning. Three days after she was missed, her body was found in a well much decomposed, but presenting no signs of delivery. It was left all day lying on the ground in the sun (in June), a woman, a relative, watching it from a little distance. No one touched the body during the day, and the watcher observed no sound or movement in it. In the evening, however, when the corpse was being lifted on to a charpoy, something was noticed hanging between its legs. At the post-mortem examination it was found that "the uterus, with all its contents, had emerged from the vagina, and was turned inside out. The contents were a full-grown male fœtus, with the remains of the membranes, funis, and placenta, all continuous with each other, but detached from the uterus." No attempt to cause abortion had been made, and no signs indicating that any such attempt had been made, were to be seen.

ILLUSTRATIVE CASES—PART D.

(See Chapters XXXIV. and XXXV.)

Insanity.

1. Illusions at first spectral only, afterwards accompanied by illusions of hearing.—Nicolai, an Academician and bookseller at Berlin, in a paper read by himself before the Royal Society of that city in 1799, gave an account of illusions from which he had suffered, of which the following is an abstract. After a period of mental disquietude consequent on a quarrel, he began to see figures which he was conscious were illusions. There appeared to him the figure of a deceased person which stood about ten yards off, and remained for about eight minutes; this in about two hours was succeeded by other phantoms. After a day or two, more figures appeared. There was no connection betwixt the phantoms and his thoughts, nor could he produce at will spectral figures of his friends. Nicolai had no difficulty in distinguishing these figures from living persons, and was fully conscious that they were illusions, the extraordinary consequences of indisposition. In about four weeks after their first appearance, says Nicolai, "the number of these apparitions increased. I began to hear them speak; sometimes they spoke to each other, generally to me. Their discourse was agreeable and short. Occasionally I took them for sensible and tender friends who strove to soften my grief." After an application of leeches to the head the figures all disappeared, nor had they returned up to the time of reading the paper eight years afterwards.

2. Illusions at first spectral only, afterwards accompanied by an illusion of hearing and sensation. (Case of Mr. H., Guy's 'Factors of the Unsound Mind,' p. 10.)—Mr. H. was daily in the habit of seeing spectral illusions. One night he retired to his bedroom with his mind full of the death of a friend which had occurred about three years previously under very distressing circumstances. He undressed, put out his candle, but had no sooner done so, than he felt his arm suddenly grasped a little below the shoulder, and forcibly pressed to his side. He struggled and called out, "Let go my arm," when he distinctly heard the words, "Don't be afraid," uttered in a low tone. He asked leave to light the candle, and felt his arm released. He lighted his candle, and then turning towards the door saw the figure of his deceased friend standing before him. It was indistinct and hazy,

but perfectly defined. Of this illusion, and indeed of the many others to which he was subject, Mr. H. said that "he never for a moment could have considered it a real object."

3. Somnambulism. (Browne's 'Medical Jurisprudence of Insanity, p. 237.)—"A butcher's boy, about sixteen years old, apparently in perfect health, after dozing a few minutes in his chair, suddenly started up, and began to employ himself about his usual avocations. He had saddled and mounted his horse, and it was with the greatest difficulty that those around him could remove him from the saddle and carry him within doors. While he was held in the chair by force, he continued violently the actions of kicking, whipping, and spurring. His observations regarding orders from his master's customers, the payment at the turnpike gate, &c., were seemingly rational. The eyes, when opened, were perfectly sensible to light. It appears that flagellation even had no effect in restoring the patient to a proper sense of his condition. The pulse in this case was 130 full and hard. On the abstraction of thirty ounces of blood, it sunk to eighty, and diaphoresis ensued. After labouring under this frenzy for the space of an hour he became sensible, was astonished at what he was told had happened, and stated that he recollected nothing subsequent to his having fetched some water, and moved from one chair to another, which indeed he had done immediately before his delirium came on."

4. Somnambulism influenced by external impressions. (Carpenter's 'Mental Physiology,' p. 600.)—Case of an officer who served in the expedition to Louisburgh in 1758. "The course of this individual's dreams could be completely directed by whispering into his ear, especially if this was done by a friend with whose voice he was familiar, and his companions in the transport were in the constant habit of amusing themselves at his expense. At one time they conducted him through the whole progress of a quarrel which ended in a duel; and when the parties were supposed to be met, a pistol was put in his hand, which he fired and was awakened by the report. On another occasion they found him asleep on the top of a locker in the cabin, when they made him believe he had fallen overboard, and exhorted him to save himself by swimming. He immediately imitated all the motions of swimming. They then told him that a shark was pursuing him, and entreated him to dive for his life. He instantly did so with such force as to throw himself entirely from the locker upon the cabin floor, by which he was much bruised and awakened of course." After these experiments he had no distinct recollection of his dreams, but only a confused feeling of oppression and fatigue.

5. The act of stabbing performed during sleep. (Taylor, ii. p. 600.)—"Two persons who had been hunting during the day slept together at night. One of them was renewing the chase in

his dream, and imagining himself present at the death of the stag, cried out, 'I'll kill him! I'll kill him!' The other, awakened by the noise, got out of bed, and by the light of the moon beheld the sleeper give several deadly stabs with a knife on that part of the bed which he had just quitted."

6. A man stabbed by his brother under similar circumstances. (Browne's 'Medical Jurisprudence of Insanity,' p. 241.)—A Spaniard, æt. twenty-six, who had been a soldier, always of good conduct, and in tolerable health, was subject every spring to epistaxis, also to talking in his sleep. The spring of 1854 passed without epistaxis, and from this time, particularly during the night, he was subject to certain moral disturbance for which purging was advised. Travelling with a brother, and sleeping in the same bed, he was attacked during the night by this excitement, fancied that his bedfellow was going to kill him, and seizing a knife he plunged it into his neck. He then went out and slept on the staircase two hours. When he awoke he had some obscure consciousness of what he had done, and on seeing his dead brother, he was in despair, and wounded himself severely. The flow of blood restored his reason, and he called for help, and after some time told all the circumstances. The man was tried for the murder, but was acquitted on the medical evidence.

7. A man suddenly aroused from sleep stabs another (*Ib.* p. 241).—"A pedlar, who was in the habit of walking about the country armed with a swordstick, was awakened one evening, while lying asleep on the high road, by a man suddenly seizing him, and shaking him by the shoulders. The man, who was walking by with some companions, had done this out of a joke. The pedlar suddenly awoke, drew his sword and stabbed the man, who soon afterwards died. He was tried for manslaughter, and, although his irresponsibility was strongly urged by his counsel, was convicted."

8. Higher form of Somnambulism. (Carpenter's 'Mental Physiology,' p. 593.)—An eminent Scottish lawyer had been consulted about a case of great difficulty and importance, and had been studying it closely and anxiously for several days. One night his wife saw him rise from his bed and go to a writing-desk which stood in his bedroom. He then sat down and wrote a long paper, which he carefully put by in his desk, and returned to bed. The following morning he told his wife that he had had a most interesting dream; that he had dreamt of delivering a clear luminous opinion respecting a case which had exceedingly perplexed him, and that he would give anything to recover the train of thought which had passed before him in his dream. She then directed him to his writing-desk, where he found the opinion clearly and fully written out."

9. Higher form of Somnambulism. (Guy's 'Factors of the Unsound Mind,' p. 71.)—A banking house once gave to a Dutch

professor of mathematics (Professor Van Swinden, of Amsterdam) a question to solve which required a long and difficult calculation. He first tried it himself several times, but never without mistake; so he handed it over to ten of his pupils. One of these attacked the problem with great vigour, but more than once without success. Late in the night which preceded the day fixed for the giving in of the answers, he went to bed baffled and tired. But in the morning, most strange to relate, he finds a paper on his desk, in his own handwriting, on which the problem is solved, without a single blunder. He had calculated the problem in his sleep, and in the dark. It was singularly clear and condensed, and the professor himself declared that he had never thought of a solution so simple and concise."

10. Intellectual imbecility. (Guy, p. 182.)—"A man of forty, of weak intellect from birth, but capable of such education as fitted him for the post of a copying clerk. He fell into bad company, committed theft, and was tried and acquitted on the ground of insanity. In general he is quiet, inoffensive, and taciturn, but answers simple questions rationally. He is subject to frequent attacks of excitement, preceded by shuffling of the feet. In these attacks, which last several days, he talks incoherently, is restless, and will strike and kick those about him. When he was about thirty years old he shut the door of his room, placed a long form close to the fire, laid himself on the form, and his head on the grate. He was found insensible, but, on being removed to an open window, had copious bleeding from the nose, and soon came to his senses. His head was burnt to the bone."

11. Intellectual imbecility. (Guy's 'Factors of the Unsound Mind,' p. 173.)—John Barclay was tried and executed at Glasgow, in 1833, for the murder of one Samuel Neilson. Barclay had shown some affection for his victim, but killed him that he might possess himself of three one-pound notes and a watch, which he took from him. After the murder, Barclay hovered about almost without disguise, and while going to spend part of the money with the first person he met, dropped first one and then another note at his feet. When questioned, he could see no difference between killing a man and killing an ox, except that he "would never hear him fiddle again," and he looked on the watch as an animal, and when it stopped, thought it had died of cold from the glass being broken. In his parish he was known as "daft Jock Barclay," and the clergyman, who knew him well, "always regarded him as imbecile, and had never been able to give him any religious instruction, and did not consider him a responsible being."

12. Moral imbecility. (Browne's 'Medical Jurisprudence of Insanity,' p. 71.)—Cuthbert Carr gave himself up to the police, confessing to the murder of a female child, aged six. By his own voluntary and detailed confession he choked the child while he

was having connection with her, to prevent her informing against him. He had been attacked with venereal disease, and his object in having connection with the child was to cure himself. After the murder he showed great shrewdness in the measures he adopted to avert suspicion. In his confession he stated that he knew doctors could not, or would not, cure the disease; that they did their best to protract a disease, and, when they could not protract it any longer, killed their patients; that they poisoned the wells in cholera time, &c., &c. Dr. Browne reported that he found him to be labouring under mental weakness or defect, probably congenital, and that his general appearance and manners were such as are usually associated with partial mental defect or eccentricity. That otherwise he was of fully average intelligence, expressing himself with accuracy and facility, that his powers of calculation and memory were unusually acute, and that he was perfectly capable of distinguishing between right and wrong. He was acquitted, on the ground of insanity.

13. The Windham Case. (Browne, *ib.*, p. 67.)—In this case W. F. Windham was alleged to be of unsound mind, and incapable of managing his affairs. It was proved that he had been sent to Eton, but that he had profited very little by the means of education which were placed in his power. He was wholly unlike other boys, and when he came of age, in 1861, his conduct was such as to lead to a belief, in the minds of those who were acquainted with his position, that he was insane. It was further proved that he was utterly deficient in business capacity; that he was extravagant in purchasing articles which he did not require, at exorbitant prices and in unnecessary quantities; that in consequence of such acts he incurred enormous debts, without having any reasonable prospect of being able to meet the demands when they became due; that he was guilty of gross indecency of language and conduct in public places, and that even the presence of ladies was not a restraint; that his appetite was voracious; that he associated constantly with people of the most indifferent character; that three weeks after he came of age he married a woman of disreputable character and life; that he married her knowing that up to the night previous to the marriage she had lived with one of his friends as his mistress; that having married her, he infected her with the venereal disease, and subsequently presented her with jewellery of the value of from £12,000 to £14,000, and settled £800 a year on her for life; that his income, at the time he did this, was not more than £1,500 per annum. The evidence went further to show that although his wife, subsequent to her marriage, cohabited with another man, Mr. Windham condoned this act by residing with her even after her adultery; that he was in the habit of acting as a railway guard, was careless as to personal cleanliness, and on occasion displayed an utter want of feeling. There was great conflict of medical

testimony in this case, but the result was that the jury, by a majority, returned a verdict that Mr. Windham was of sound mind, and capable of taking care of himself and his affairs.

14. An imbecile greatly under the dominion of a childish fancy. (Guy's 'Forensic Medicine,' p 185.)—"A young gentleman, aged twenty, was the slave of a childish fancy for windmills, with an aversion equally strong to water-mills. Having been placed under control in a place where there were no windmills, he cut the calves of a child's legs through to the bone, and stated that he would have taken away its life, that he might be tried for his act and removed from a place where there were no windmills. He had always been violent when thwarted in his fancy, had threatened his keepers and members of his family, and had more than once made preparations for committing murder."

15. Deficient sensibility to pain in general intellectual mania. (Taylor, ii. p. 475.)—A maniac, during violent exertion, met with a severe compound fracture of the leg. The fracture was carefully put up by the medical attendants, the maniac watching the whole proceeding attentively. In a day or two symptoms of great constitutional disturbance showed themselves, although apparently the injured limb was progressing satisfactorily. Accordingly the surgeons proposed removing the splints, and were preparing to do so, when the maniac suddenly drew the fractured leg out of a hole in the mattress, in which he had concealed it, and held it up before them. It was then discovered that the patient, soon after the fracture had been put up, had removed the splints from the injured limb, and applied them, with great nicety, to the sound one. Although the injured leg was inflamed, the bones displaced, and the wound filled with feathers, the patient had not expressed any feeling of pain.

16. Death of a lunatic from injuries received during a struggle with his attendants. (Taylor, ii. p. 475. *Reg.* v. *Slater and Vivian,* C. C. C. Sept. 1860.)—The prisoners were attendants in an asylum, and were tried for the manslaughter of a patient. The evidence showed that the deceased died rather suddenly three days after a serious struggle with one of his attendants. On post-mortem examination eleven ribs were found fractured; there was, however, no displacement. Slight lacerations of the left lobe of the liver were also present. Two insane patients alleged that deceased had been greatly maltreated by the prisoners shortly before death. The question therefore arose, when were the injuries inflicted. On the one hand it was represented that if the injuries had been inflicted during the struggle three days before death, marked symptoms would have been present during the interval between the receipt of the injuries and death, and that as no such symptoms were present, the story told by the two lunatics was probably true. On the other hand the medical officers of the asylum considered it possible that the

injuries which caused death were inflicted in the struggle three days previously, and that the absence of symptoms was accounted for by insensibility to pain, especially as there was incipient general paralysis. The accused were acquitted

17. Monomania of persecution; multiple delusions connected with one morbid idea. (Bucknill and Tuke, p. 219.)—A female patient was "perfectly convinced of the existence of a persecuting fellow in a room above her own, who vented all his malignity upon her by means of certain machinery and wires . . . He 'brays' her in the night with three of these wires, so that she is stiff in the morning, and covered with marks as if she had been switched . . . At other times he will thrust three wires into her mouth, which leave 'a very bitter verdigris *taste*' therein. She protests that she can see a 'hole like the cut of a knife' in one corner of the ceiling, through which he introduces the wires . . . She has stopped her clock and covered it up, because he used to employ his wires to make it strike some twenty times in the night, in order to disturb her." He also, she believes, delights in sending her to sleep with chloroform, which she feels dropping from the ceiling upon her cap.

18. Religious monomania. Homicide. (*Reg.* v. *Wilson*, Lincoln Summer Assizes, 1864: Taylor, ii. p. 554.)—"A woman consulted a medical man as to pains in her head, loss of appetite, and low spirits after her delivery; she was also suffering from religious despondency. While in this state, she got up in the night and drowned four of her children in a cistern. She gave this account of the act:—She washed the children, put them to bed, and retired herself about 10 o'clock, but could not sleep: and between 12 and 1 o'clock it was suggested to her mind, as she says, by a black shadowy figure, that if they were in heaven they would be out of danger and better done to than she could do for them. It was still further suggested to her mind in the same way, that she could easily put them into the cistern, and she at once proceeded to do so; it was better for them to die young than to grow up wicked."

19. Monomania of persecution. Homicide. (Maudsley's 'Phys. and Path. of the Mind,' p. 371.)—"A young man, who had previously had a few epileptic fits, became extremely melancholic. Being possessed with the idea that he was to be murdered in his father's house, he made frequent attempts to escape from it . . . His father was a butcher; and the young man becoming calmer after a time, and being thought trustworthy, was permitted at his own request to be present at the slaughter of an ox, but when all was finished, he did not wish to return home. His friends, however, pressed him, and two of them taking him by the arm in a friendly manner, accompanied him towards his home; but just as he approached the door of his house he suddenly drew out a butcher's knife, which he had concealed, and stabbed to the

heart one of them; fleeing immediately to the forest, where he passed the night. Next morning he went to the house of a relative, who lived some distance off, and said that he had run away from home, as they wished to kill him there."

20. Monomania of persecution detected with difficulty. (Carpenter's 'Mental Physiology,' p. 669.)—"Dr. A. T. Thomson was requested to see a gentleman, whose friends were desirous of placing him under restraint, being well assured of his insanity from the supervention of uncontrollable outbreaks of temper, to which he had never previously given way, though they could find no ostensible ground in his conversation or actions which would legally justify the use of coercive measures. Several medical men had been consulted, all of whom had failed to obtain any such justification . . . Dr. Thomson, struck with the evidence of violent passion, afforded by the damages done to the furniture of this gentleman's apartments, 'felt convinced that there was some perversion of feelings or intellect which it was his business to discover.' For two hours he conversed with his patient on a variety of topics, and never enjoyed a more agreeable or instructive conversation, his patient being evidently a man of great attainments. Dr. Thomson was beginning to despair of finding out the mystery of his disorder, when it chanced that animal magnetism was adverted to, on which the patient began to speak of an influence which some of his relatives had acquired over him by this agency, described in the most vehement language the suffering he endured through these means, and vowed vengeance against his persecutors with such terrible excitement, that it was obviously necessary, alike for their security and his own welfare, that he should be placed under restraint."

21. Monomania; the delusion only discoverable with difficulty. (Case related by Erskine during his defence of Hadfield, tried for shooting at George III.: Browne, p. 290.)—"A person, who had been confined in a lunatic asylum, prosecuted his brother and the keeper of the asylum for false imprisonment and duress Erskine was informed that the man was undoubtedly insane, but was not told the particular form which the malady assumed. The prosecutor, himself a witness in support of the indictment, was put into the witness-box and examined; and when Erskine came to cross-examine him, he found his evidence clear, distinct, collected, and rational. He tried to discover some alienation of mind; but during a cross-examination, conducted with all the skill and sagacity of which he was master, for nearly an hour, he was completely foiled; the answers were perfectly rational—there was not the slightest sign of mental alienation. A gentleman, however, who had been accidentally detained, came into Court, and whispered in Erskine's ear that the witness thought he was the Saviour of mankind. On receiving the hint,

Erskine made a low bow to the witness, addressed him in terms of great reverence, and respectfully begged to apologise for the unceremonious manner in which he had treated a person of his sacred character, and called him by the name of Christ. The man immediately said, 'Thou hast spoken truly; I am the Christ.'"

22. Monomania readily controlled. (Guy's 'Forensic Medicine,' p. 208.)—"Henry Weber, Sir Walter Scott's private secretary, became addicted to habits of intoxication, which injured his health. One evening, Scott, after Weber's return from Edinburgh, observed Weber's eye fixed upon him with unusual solemnity of expression. On inquiring after his health, Weber rose and said, 'Mr. Scott, you have long insulted me, and I can bear it no longer. I have brought a pair of pistols with me, and must insist on your taking one instantly,' and with that he produced the weapons and laid one of them on Scott's manuscript. 'You are mistaken, I think,' said Scott, 'in your way of setting about this affair; but no matter. It can, however, be no part of your object to annoy Mrs. Scott and the children; therefore, if you please, we will put the pistols in the drawer until after dinner and then arrange to go out like gentlemen.' Weber answered with equal coolness, 'I believe that will be better,' and laid the second pistol also on the table. Scott locked them both up in his desk, and said, 'I am glad you feel the propriety of what I suggested; let me only request further that nothing may occur while we are at dinner to give my wife any suspicion of what has been passing.' Weber again assented, and Scott withdrew to his dressing room, dispatched a message to one of Weber's intimate companions, and had the maniac secured and placed in confinement."

23. General moral mania. (Browne, p. 114.)—"W. R. was 27 years of age. He had been eight times in the House of Correction. His father was an epileptic, and he himself had been subject to convulsions when teething, and at intervals during his after-life. He tortured animals, picked out the eyes of a kitten with a fork. He lied and stole. He was expelled from school as too bad to be kept. He afterwards consorted with the worst characters; was drunken, debauched, dishonest. He attempted or pretended to commit suicide. He was utterly false and untrustworthy. He delighted in torturing those patients who were, like himself, confined in the lunatic asylum, and who were too weak to resent injury with violence. He was indelicate in the presence of females, and attempted a rape on his mother and on his sister. Yet with all he was intelligent, exceedingly cunning, and, while actually the victim of epileptic seizures, he was prone to feign fits, and did it with considerable ability. In spite of careful watching he repeatedly effected his escape; was exceedingly vain, and in the presence of some persons seemed to be ex-

ceedingly devout. He was ingenious in excusing his errors, and, although exceedingly mischievous, was careful to avoid disagreeable consequences." This individual, Browne further remarks, was possessed of "an intelligence of such high order as to enable him thoroughly to understand the relation between a found-out crime and its punishment, for he invariably tried to conceal the commission of the criminal act by lies, hypocrisy, and various clever explanations."

24. General moral mania. (Maudsley, 'Phys. and Path. of the Mind,' p. 362.)—"An old man, aged 69, who had been in one asylum or another for the last fifteen years of his life. He had great intellectual power, could compose well, write tolerable poetry with much fluency, and was an excellent keeper of accounts. There was no delusion of any kind, and yet he was the most hopeless and trying of mortals to deal with. Morally he was utterly depraved; he would steal and hide whatever he could, and several times made his escape from the asylum with marvellous ingenuity. He then pawned what he had stolen, begged, and lied with such plausibility that he deceived many people, until he finally got into the hands of the police, or was discovered in a most wretched state in the company of the lowest mortals in the lowest part of the town. In the earlier part of his insane career, which began when he was 48 years old, he was several times in prison for stealing. In the asylum he was a most troublesome patient; he could make excellent suggestions, and write out admirable rules for its management, and was very acute in detecting any negligence or abuse on the part of the attendants when they displeased him, but he was always on the watch to evade the regulations of the house, and when detected, he was most abusive, foul, and blasphemous in his language. He was something of an artist, and delighted to draw abominable pictures of naked men and women, and to exhibit them to those patients who were addicted to self-abuse. He could not be trusted with female patients, for he would attempt to take indecent liberties with the most demented creature. In short, he had no moral sense whatever; while all the fault that could be found with his very acute intellect was that it was entirely engaged in the service of his depravity . . . At long intervals, sometimes of two years, this patient became profoundly melancholic for two or three months, refused to take food, and was as plainly insane as any patient in the asylum. It was in an attack of this sort also that his disease first commenced."

25. Homicidal mania, gradual approach. (Bucknill and Tuke, p. 268.)—"A young man, æt. 25, and of gentlemanly appearance, after giving his address, and declaring himself to be a schoolmaster in a certain well-known college (in Paris), begged that the Commissary of Police would take him in charge with a view to his confinement in the Asylum of St. Ann. He then explained

that he was not mad in every respect; on the contrary, he possessed the full use of his mind, only while sleeping among the pupils confided to his charge, he was seized with the most destructive inclinations. Night after night in an agony of fear he had struggled with himself, and it was with the greatest difficulty that so far he had succeeded in restraining his intense desire to strangle one or two of the little boys. Now all his energies were exhausted. He felt that this unknown power would ultimately triumph over him, and rather than commit the crime he placed himself in the hands of the police. At this moment a boy accused of theft was brought into the room. The eyes of the schoolmaster were immediately lit with a strange light; and had it not been for the timely assistance of a brawny policeman, the boy would have been throttled before the eyes of justice."

26. Homicidal mania; previous symptoms of mental disorder slight only. (*Reg.* v. *Brixey*, Taylor, ii. 564.)—"The prisoner, a quiet, inoffensive girl, a maidservant in a respectable family, was charged with the murder of an infant. She had laboured under disordered menstruation, and a short time before the occurrence had shown some violence of temper about trivial domestic matters." This was all the evidence of insanity exhibited previous to the act. "She procured a knife from the kitchen on some slight pretence, and, while the nurse was out of the room, cut the throat of her master's infant child. She then went downstairs and told her master what she had done. She was perfectly conscious of the act she had committed; she treated it as a crime, and showed much anxiety to know whether she should be hanged or transported. There was not the slightest evidence that at the time of the act, or at any time previously, she had laboured under any delusion, or intellectual aberration. The prisoner was acquitted on the ground of insanity, probably arising from obstructed menstruation."

27. Homicidal mania co-existing with a quiet exterior. (Taylor's Manual, p. 743.)—A Commissioner of Lunacy, deeply impressed with the conviction that a patient in the asylum which he was visiting was perfectly sane, and fit to be discharged, in defiance of the physician's warning, trusted himself alone in the lunatic's company for the purpose, as he said, of a private conversation. In less than five minutes after they were alone, and as the lunatic believed unobserved, the Commissioner was throttled by his companion, and but for the timely intervention of the physician, who had been a secret spectator of the scene, would have been strangled.

28. Homicidal mania in an individual otherwise apparently sane. (Guy's 'Factors of the Unsound Mind,' p. 181.)—"William Brown strangled a child whom he met by accident, and then requested to be taken into custody. On the trial he

said he had never seen the child before, and had no malice against it, and could assign no motive for the dreadful act. He bore an exemplary character, and had never been suspected of being insane."

29. Kleptomania. (Browne, p. 128.)—"Mr. M—— was an individual of high rank. He was the owner of an excellent estate, and was as wealthy as most of his neighbours in the county in which he resided. He was never suspected of being insane, and the only evidence of mental unsoundness that could have been obtained was a confession on the part of some of his servants that he was 'sometimes peculiar.' Yet this gentleman was in the habit of appropriating 'towels.' He invariably, when visiting or on a journey, packed the towels he found in his bedroom in his portmanteau. And when he returned home, the stolen articles were, by his own directions, returned to their real owners."

30. Alleged Kleptomania. (Casper, iv. p. 308.)—Frau von X—, a lady of a certain rank, committed during her pregnancy theft in three goldsmiths' shops. Apparently while committing the first theft she was surprised by the assistant in the shop, on which she became pale, asked for a glass of water, and went away hastily, scarcely drinking any. Three months afterwards, and four weeks before her confinement, she went to a second and third shop, making small purchases, and offering in payment fragments of articles stolen from the first shop. At one of these shops she accepted ten thalers (thirty shillings) in cash, in part payment for the articles she offered. She concealed her conduct from her husband until she was summoned after her delivery, when she confessed to him her thefts, accounting for them by stating that during her pregnancy she had been seized with an irresistible desire to possess herself of glittering objects. She also said that she had gone out with the intention of returning the articles she had taken, but had become convinced by the way that they were her own property, properly acquired. Much evidence was given which went to show the existence of mental aberration. Casper being referred to gave it as his opinion that Frau von X—— was criminally responsible, that in fact in her case the propensity to acquire was not irresistible, and gave as reasons: "1. That although the accused had besought her husband not to take her to those places where shining objects were to be seen, she went to goldsmiths' shops of her own accord, and without any necessity for doing so. 2. That she had paid away silver. 3. That she broke up the objects she stole in order that they might not be recognised, and in that way lead to her detection. 4. She had not gone to the same goldsmith's shop twice. 5. She had concealed her conduct from her husband; and 6. When interrogated she had made many false and contradictory statements." (Browne, p. 138.)

31. Homicidal propensity in an imbecile. (Guy's 'Factors of

the Unsound Mind,' p. 174.)—A. H., æt. 28, was tried for the murder of a boy æt. 13, at Taunton Assizes, in March 1868. The prisoner was notoriously an imbecile from his childhood, but was considered by his father, an officer in the army, and others, to be quiet, well disposed, and harmless. He was a diligent reader of the Bible, and had expressed a wish to be a Scripture reader. He was greatly addicted to wandering about from place to place. On the morning of the murder he walked twenty miles, armed with a big stick and a sharp knife. Early in the evening of this day, he gave himself up to the police as "having killed a human being," and crying bitterly at the thought of what he had done. He had been tempted to kill two or three persons on the road, but had abstained. At length he killed a "poor little boy in a field," afterwards washing the blood from his hands. He conducted the parish constable to the spot where his victim lay, the head beaten to a pulp, and nearly severed from the trunk by a wound in the throat extending from ear to ear. He could not bear to look at the body. He stated that he had been reading of murders in the newspapers till he thought he must commit one; that the thought had been in his mind for a week; that he was compelled to do it; that he did not wish to kill the boy, but could not resist it; that he knew he was doing wrong, but had no power to resist. The prisoner was acquitted on the ground of insanity.

32. Insanity due to anxiety of mind caused by a criminal charge. (Taylor, ii. p. 496.)—A poor man, a shoemaker, was requested by two police-officers to assist them in conveying to prison two men committed on a charge of theft. The shoemaker took a gun with him, and on the order of the police-officers fired at one of the prisoners, who was attempting to escape, and wounded him severely. The shoemaker was committed to gaol as a criminal, and the event made "such an impression upon him that he became violently maniacal."

33. Feigned insanity, silly answers to questions. (Woodman and Tidy, p. 900, quoted from the Berlin Medical Zeitung.)—A widow, who had bought a house, and not liking it, wished to annul the contract, feigned insanity. When asked to count, she did so thus: 1, 2, 4, 6, 7, 8, 10, 11, 13, &c. Asked how many fingers she had on each hand, she said "four." Asked how many two and two made, she said "six." To some simple questions such as—How many children have you? How long has your husband been dead? What did he die of? What is your daughter's name? What have you had to eat to-day? What is your clergyman's name?—she in each case gave an incorrect answer. To other simple questions, such as—What year is this? How long is it since Christmas? Where do you live? &c., her answer was, "I don't know." Asked what is the first commandment, she answered, "I am the Lord thy God." Asked what is the second, she gave the

same answer; said she did not know the third and fourth. Asked the fifth, she said, "Thou shalt *not* honour thy father and mother."

34. Acts apparently inexplicable the result of a delusion. (Guy's 'Forensic Medicine,' p. 206; quotation from the Autobiography of a Religious Maniac.)—"I expected to be guided to prayer, but a spirit guided me and placed me in a chair in a constrained position, with my head turned to look at the clock, the hand of which I saw proceeding to the first quarter; I understood I was to leave the position when it came to the quarter. Another delusion I laboured under was that I should keep my head and heart together, and so serve the Lord, by throwing myself with precision and decision head over heels over every stile or gate I came to."

35. Homicide by an insane; forethought and preparation shown. (Guy's 'Forensic Medicine,' p. 208.)—"A patient confined in the Manchester Lunatic Asylum had been cruelly treated by a keeper, and in revenge killed him. He related the particulars of the transaction to Dr. Haslam with great calmness and self-possession. He said, 'The man whom I stabbed richly deserved it. He behaved to me with great violence and cruelty.' After detailing the treatment, he went on, 'I gave him warning; for I told his wife I would have justice of him. On her communicating this to him he came to me in a furious passion, threw me down, dragged me through the courtyard, thumped me on my breast, and confined me in a dark and damp cell. Not liking this situation, I was induced to play the hypocrite. I pretended extreme sorrow for having threatened him, and by an affectation of repentance, prevailed on him to release me. For several days I paid him great attention and lent him every assistance. He seemed much pleased with the flattery, and became very friendly in his behaviour towards me. Going one day in the kitchen, where his wife was busied, I saw a knife; this was too great a temptation to be resisted. I concealed it about my person and carried it with me. For some time afterwards the same friendly intercourse was maintained between us; but as he was one day unlocking his garden door, I seized the opportunity and plunged the knife up to the hilt in his back.'"

36. The McNaughten case. (Maudsley's 'Med. Jurisp. of Insanity,' p. 95.)—In this case, a man, named McNaughten, was tried for the murder of a Mr. Drummond, and acquitted on the ground of insanity. McNaughten was under a delusion that Drummond was one of a number of persons whom he believed to be following him everywhere, blasting his character, and making his life wretched. Under the influence of this delusion he shot Drummond. McNaughten had transacted business a short time before the deed, and had shown no obvious symptoms of insanity in his ordinary discourse and conduct.

37. Validity of a will made by an insane. (*Banks* v.

Goodfellow, L. R., 5 Q. B. 549: Browne, p. 191, and Maudsley, 'Responsibility in Mental Disease,' p. 117.)—Lord Cockburn, in delivering judgment in this case, said: "It is essential to the exercise of such a power that a testator should understand the nature of the act and its effects; shall understand the extent of the property of which he is disposing; shall be able to comprehend and appreciate the claims to which he ought to give effect; and, with a view to the latter object, that no disorder of the mind should poison his affections, pervert his sense of right, or prevent the exercise of the natural faculties; that no insane delusion shall influence his will in disposing of his property, and bring about a disposal of it which, if the mind had been sound, would not have been made." . . . "But, when in the result the jury are satisfied that the delusion has not affected the general faculties of the mind, and can have had no effect upon the will, we see no sufficient reason why the testator should be held to have lost his right to make a will, or why a will made under such circumstances should not be upheld." . . . "In the case before us two delusions disturbed the mind of the testator—the one, that he was pursued by spirits; the other, that a man, long since dead, came personally to molest him. Neither of these delusions—the dead man not having been in any way connected with him—had or could have had any influence upon him in disposing of his property. Under these circumstances, then, we see no ground for holding the will to be invalid."

38. Testamentary incapacity. (*Smee and Others* v. *The Corporation of Brighton*, L. R., P. D. 5, p. 84.)—In this case two wills were propounded, one made in 1859 and the other in 1867. By the first the testator left nearly the whole of his property to his wife absolutely. By the second he gave it her for her life or widowhood only, after which it was to go to the Corporation of Brighton for the purpose of forming a public library in the Royal Pavilion there. It was proved that at the time the wills were made the testator laboured under various delusions, the chief of which were that he was a son of George IV., that when he was born a large sum of money was placed in his father's hands in trust for him, and that his father had robbed him of part of this and had given it to his brothers. Sir James Hannen, in summing up the case to the jury, said, "You should specially bear in mind that any one who questions the validity of a will is entitled to put the person who alleges that it was made by a capable testator upon proof that he was of sound mind at the time of its execution. The burden of proof rests upon those who set up the will. . . . You have, therefore, to be satisfied from the evidence which has been offered by those propounding the will of 1867 and the earlier will also, that the delusions under which the deceased laboured were of such a character that they could not reasonably be supposed to affect the disposition of his property.

This is an extremely delicate and difficult investigation, and may be illustrated by reference to the physical world. There might be a little crack in some geological stratum of no importance in itself, and nothing more than a chink through which the water filters into the earth; but it might be shown that this flaw had a direct influence upon the volume, or colour or chemical qualities of a stream that issued from the earth many miles away. So with the mind. Upon the surface all may be perfectly clear, and a man may be able to transact ordinary business or follow his professional calling, and yet there may be some idea through which, in the recesses of his mind, an influence is produced on his conduct in other matters." After pointing out that George IV. had taken a great interest in the town of Brighton, and that the testator was under the delusion that his brothers by the fraud of his father had been put in possession of two-thirds of his (the testator's) property, Sir James Hannen concluded thus: "It being conceded that the testator was undoubtedly of unsound mind, are you satisfied that when he made either of the wills he was capable of dealing with the subjects before him entirely free from the delusions under which he suffered? If the evidence does not satisfy you as to this, your verdict should be against the will." The jury found against both wills.

39. Question of insanity in regard to validity of marriage. (*D.* v. *D.*, otherwise *M.*, "Times," March 11th, 1885.)—In giving judgment in this case, Sir James Hannen said: "The question I have to determine is whether the respondent at the time of her marriage, on the 28th October, 1882, was of sound mind, so as to be able to enter into the contract of matrimony. . . . I am of opinion that every case of this kind must be decided on its own facts. . . . I accept for the purposes of this case the definition (of soundness of mind) which has been substantially agreed upon by the counsel . . . namely, a capacity to understand the nature of the contract and the duties and responsibilities which it creates. It is to be observed, however, that this only conceals for a moment the difficulties of the inquiry, for we have still to determine the meaning to be attached to the word 'understand.' If I were to attempt to analyse this expression, I should encounter the same difficulties at some other stage of the investigation with reference to some other phrase, and I should still have to determine on the review of the whole facts, whether the respondent came up to the standard of sanity, which I must fix on in my own mind, though I may not be able to express it. I may say this much at the outset, that it appears to me that the contract of marriage is a very simple one, which does not require a high degree of intelligence to comprehend it. . . . I agree with the Solicitor-General (for the plaintiff) that a mere comprehension of the meaning of the words of the promises exchanged

is not sufficient. The mind of one of the parties may be capable of understanding the language used, but may yet be affected by such delusions, or other symptoms of insanity, as may satisfy the tribunal that there was not a real appreciation of the engagement entered into."

40. Undue influence affecting the validity of the marriage of an insane. (Woodman and Tidy, p. 890, and Abercrombie, p. 169. Case of the Earl of Portsmouth.)—"In the suit for the dissolution of the marriage of the Earl of Portsmouth, on the ground that he was of weak, and afterwards of unsound mind, it was proved that his servants were his playfellows, and that he was fond of driving carts loaded with dung or hay, that he was occasionally extremely cruel to his horses and domestics, &c., &c. He was, although of age, in the hands of guardians. One of these, a solicitor, persuaded him to marry his daughter, without communicating with the relations or other guardians, and the marriage was afterwards declared void on account of the undue influence used."

41. Legal justification of restraint. (*Neave* v. *Heatherley*, Q. B. D., August 1885.)—In this case Lord Coleridge, C.J., said that the examination of a person previous to placing him in an asylum ought to be "a real inquiry, a real weighing and sifting of evidence, a real examination, a real serious and solemn exercise of judgment," in order to ascertain whether an individual came within the definition of the statute of "a lunatic, idiot, or person of unsound mind, and a proper person to be taken charge of and detained under treatment. . . . He emphatically dissented from the Attorney-General (for the plaintiff) that unless every other means had first been exhausted, a person ought not to be placed in an asylum. The abuse of a thing was no proof that it had not a use, and early treatment in cases of unsoundness of mind was of the very greatest importance. People living in small houses had no power of making provision for such early treatment of relations who might be unsound in mind, while relegation, at an early stage, to a well-appointed asylum was calculated to have the best results."

42. Negligence in filling up a certificate of lunacy. (*Hall* v. *Semple*, 3 F. & F. 337.)—In this case the plaintiff had been discharged from an asylum on the ground of informality in the certificate. This certificate was dated July 29th, but the visit and examination were made on June 13th. The defendant was one of the medical men who had signed a certificate of the plaintiff's insanity. The evidence, however, went to show that Hall, although a very bad-tempered man, was not really insane, and that the defendant had relied too much on the statements of the wife and other interested persons. Compton, J., in summing up the case to the jury, said, "The principal questions to which I desire to direct your attention are these: first, whether you

think that he (the defendant) signed the certificate untrue in fact, negligently and improperly, and without making proper and sufficient inquiries. . . . It will be for your consideration what degree of care is necessary, so as to make out by the absence of it culpable negligence. It is not a mere mistake or error in judgment which would amount to such negligence, but you must be satisfied that there was culpable negligence. . . . And, again, you are not inquiring into an error in judgment, but whether the defendant has been guilty of that culpable negligence which I have explained and described to you—negligence in not making sufficient inquiries, the examination not having been sufficient in his own judgment." The jury found that there had been culpable negligence, and awarded the plaintiff £150 damages.

MISCELLANEOUS APPENDICES.

I.—Dr. Humphrey's Table.

Measurements at different ages (in inches).

Age.		Height.	Spine.	Circumference of skull.	Humerus.	Radius.	Hand.	Femur.	Tibia.	Foot.	Pelvis. Transverse diameter of.	Pelvis. Antero-posterior.
At birth		19	7·0	15·0	3·5	2·5	3·1	4·3	3·5	3·5	1·3	1·3
2 years (average)		27	8·5	17·7	4·7	3·6	3·1	6·2	5·1	3·0	2·2	2·2
4 to 6 years (average)		35	11·8	18·0	6·6	4·8	4·1	9·1	7·1	5·1	2·5	2·5
8 to 12 years (average)		43	12·8	18·8	8·3	6·0	5·1	11 4	9·4	6·4	3·1	3·1
15 years	Female	55	17·0	19·0	10·3	7·0	5·8	14·8	11·0	7·8	4·0	3·6
	Male	54	16·5	19·0	10·5	7·5	5·6	15·0	11·5	..	..	..
	Average	54	16·6	19·3	10·4	7·4	5·7	14·8	11·6	8·0	3·8	3·6
18 to 19 years	Female	59	19·0	19 5	11·0	8·2	6·5	16·0	12·8	8·0	5·0	4·8
	Male	59	17·5	20·4	11·0	8·5	6·3	15·0	13·0	8·0	3·9	3·8
	Average	60	18·5	19·8	11·4	8·6	6·6	15·8	13·3	8·3	4·7	4·5

II.—Average Weight, corresponding to Height, of 450 Native Male Mill and other Operatives in Bombay, chiefly Natives of the Konkan and Western Deccan.

Height in feet and inches.					Weight in lbs.
Feet.	Ins.		Feet.	Ins.	
5	0	to	5	1	95½
5	1	„	5	2	99
5	2	„	5	3	100½
5	3	„	5	4	105
5	4	„	5	5	107
5	5	„	5	6	110½

Average height of the above, 5 feet 3 inches.

The average weight of 534 prisoners discharged from the Bombay House of Correction in 1876 was 105 lbs. Lewis (Memorandum on Prison Diets) states that 103 lbs. is rather above the average weight of Bengali prisoners, and that the average weight of prisoners of the North-West Provinces and Oudh, where the men are as a rule taller and heavier than in Bengal, is under 110 lbs.

III.—Average Heights and Weights of Belgians (Quetelet).

Age.	Males.		Females.	
	Height.	Weight.	Height.	Weight.
Years.	Inches	Lbs	Inches.	Lbs.
1	27·5	19·8	27·0	18·9
2	31·0	24 2	30 7	24·2
3	34·2	27 5	33·6	27·2
4	36·6	30·8	35·9	30·5
5	38·8	34 9	38·5	33·6
6	41·2	39·1	40·7	36·7
7	43·4	43·3	42 9	39·1
8	45·7	47·5	45·5	41·8
9	48·1	51·7	47·4	46·2
10	50·1	55·4	49·2	50·8
11	52·5	59·4	50·6	56·1
12	54·5	63·8	52·7	63 8
13	56·4	72·8	55·8	71·5
14	58·6	81·6	58·1	79 8
15	61·0	90·6	58·9	88·0
16	63·0	99·8	59·8	95·7
17	64·6	109·3	61·1	102·9
18	65·2	118·5	61·6	109·5
19	65 6	126·7	61·8	114·6
20	65·8	130·9	62·0	117·0
25	66 3	145·6	..	120·5
30	66 3	144·9	..	121·6
Dr. Bridge's average heights and weights of English children in non-factory districts.				
8	46·66	55·08	46·73	52·82
9	49·21	60·02	48·63	56·53
10	51·00	65·29	50·07	61·19
11	52·87	71·01	53·66	68·00
12	54·05	75·00	54·41	75·95

IV.—Progress of Ossification after Birth (Ogston).

Age.	Points of ossification appear in	Bony union occurs between
4 months	Cornua of hyoid.	
5 „	Cornicula of hyoid.	
6 „	Anterior arch of atlas.	Alæ majores and body of sphenoid.
1 year	Lower end of humerus, heads of humerus, femur and tibia; 1st cuneiform bones.	Posterior arches and body of vertebræ; portions of the temporal bone, except styloid process
2 years	Lower ends of radius, tibia and fibula; ends of metacarpal and metatarsal bones.	
2½ „	Patella, lesser tuberosity of humerus and four smaller metacarpal bones.	

IV.—Progress of Ossification after Birth (Ogston)—*continued.*

Age.	Points of ossification appear in	Bony union occurs between
3 years.	Cuboid and large trochanter.	Odontoid and axis.
4 „	Trapezoid; 2nd and 3rd cuneiform.	Styloid process and temporal bone.
5 „	Semilunar; carpal scaphoid; head of fibula; ends of finger bones.	Rami and body of vertebra dentata.
6 „	Proximal epiphyses four smaller toes.	Rami of pubis and ischium.
7 „	Trochlea of humerus.	
7 to 9 „	Olecranon and scaphoid.	The two bony points at head of humerus.
9 „	.. ..	Three portions of os innominatum.
12 „	Pisiform.	
14 „	Neck and lesser trochanter of femur.	
15 „	Inferior angle of scapula.	Last 4 sacral vertebræ, coracoid and body of scapula.
15 to 20 „	Sternal end of clavicle, coccyx.	Shaft of femur and its epiphyses; humerus and its epiphyses.
18 to 23 „	.. ..	Sphenoid and occipital; tibia and its epiphyses; 1st and middle portions of sternum; epiphyses and body of ribs.
25 to 30 „	.. ..	First sacral vertebra and rest of sacrum.

V.—Table of Measurements of the Male and Female Pelvis (Tidy).

	Male.		Female.				
	Ins.	Lines.	Ins.	Lines.		Ins.	Lines.
Between the antero-superior spinous processes of the ilia	7	8	8	6	to	10	0
Between the middle points of the cristæ of the ilia	8	3	9	4	„	11	1
The transverse . . diameter of the abdominal strait of the true pelvis	4	6	5	0	„	5	6
„ oblique . . diameter of the abdominal strait of the true pelvis	4	5	4	5	„	5	5
„ antero-posterior diameter of the abdominal strait of the true pelvis	4	0	4	0	„	4	4
„ transverse . . diameter of the cavity of the true pelvis	4	0	4	7	„	4	8
„ oblique . . diameter of the cavity of the true pelvis	5	0	5	2	„	5	4
„ antero-posterior diameter of the cavity of the true pelvis	5	0	4	7	„	4	8
„ transverse . . diameter of the perineal strait of the true pelvis	3	0	4	0	„	4	5
„ antero-posterior diameter of the perineal strait of the true pelvis	3	3	4	4	„	5	0

VI.—Statistics of Violent Deaths.

—		Bombay, 1875.	Bengal, 1875 (Selected Circles).	Central Provinces, 1875.	Hyderabad Assigned Districts, 1875.	England and Wales (average of fifteen years).
Females living, per 1000 males		935	950	965	..	1053
Death-rate per 1000, all causes		24·1	24·8	26·3	26·9	22 4
Violent deaths per million of population—						
Suicide[1]	Males	45·9	53·7	68·7	59·5	98·2
	Females	55·8	74·8	86·3	68·1	33·8
	Total	50·6	64·0	77·3	63·6	65·2
Wounds	Males	35·2	107·4	35·0	43·8	29·1[2]
	Females	14·6	72·5	30·8	24·9	14·4
	Total	25·4	90·4	32·9	34·8	21·6
Snake-bite and wild beasts	Males	97·1	148·0	152·5	118·2	..
	Females	59·8	164·9	103·4	76·7	..
	Total	79·3	156·2	128·4	98·4	..
Other violence	Males	250·6	160·4	195·0	174·2	940·5[3]
	Females	202·5	146·6	176·5	157·2	307·8
	Total	227·8	153·6	185·9	166·1	616·0
Total, all violent causes	Males	429·1	469·5	495·3	395·8	1067·8
	Females	332·7	458·8	546·8	327·0	356·0
	Total	383·1	464·3	520·3	362·9	702·7

[1] In the five years ending 1876, the average suicide death-rate per million of population was, in Madras, males, 62·6; females, 68·5. In the North-West Provinces, males, 27·8; females, 71·6; and in Calcutta, males, 64·2; females, 104·6. —K. McLeod, "On Suicide in India." [2] Homicide. [3] Accidents of all kinds.

VII.—Method of Suicide per 1000 Suicides of each Sex.

(K. McLeod, "On Suicide in India.")

—	England and Wales, 1874 & 1875.	Bombay Presidency, 1873 to 1876	Madras Presidency, 1872 to 1876.	Punjaub (2 years, 1872 & 1876).	Calcutta (Town), 1872 to 1876.	Madras (Town), 1872 to 1876.
Males—						
Hanging	274	382	471	500	326	163
Drowning	184	456	443	174	74	623
Poison	90	91	26	184	453	86
Cuts, stabs, &c.	280	71	49	26	84	96
Gunshot	82			32	63	32
Otherwise	90	..	11	84	..	..
Females—						
Hanging	284	185	179	464	519	42
Drowning	309	767	790	354	26	937
Poison	155	37	26	84	429	21
Cuts, stabs, &c.	182	11	2	18	26	..
Gunshot	2			..	..	..
Otherwise	68	..	3	80	..	..

VIII.

Deaths by drowning in Bombay in 1883, actual number of deaths = 2713
Death-rate from all injuries per 1000 of population . . . . . 0·36
Death-rate from drowning " " . . . . . 0·18

Causes per thousand deaths from drowning—			
Suicide	Males	51	
	Females	103	
Total			154
Accident—Falls into tanks and wells .	Males	175	
	Females	275	
Total			450
Other accident	Males	188	
	Females	142	
Total			330
Unknown	Males	38	
	Females	28	
Total			66
Total . .		..	1000

IX.—Deaths by Violence in Bombay in 1875, Classed as Otherwise, *i.e.* not known to be Suicide or Accident. Actual number of Cases = 425.

Ratio per million of Population.

—	Males.	Females.	Total.
Poison	2·24	0 52	1·41
Wounds	12·61	7·62	10·23
Drowning	15·32	13·68	14·54
Total	30·17	21·82	26·18
Homicide, England and Wales, average of fifteen years . .	29·13	14·44	21·59

X.—Factors for the Calculation of the Nutritive Value of Dietaries or Articles of Food

1. When the composition of the dietary or article of food is stated in ounces of albuminates, carbohydrates, and fat, the quantity of nitrogen and carbon present can be ascertained by Parkes' factors as follows:—

1 oz dry albuminate contains . . . . . 69 grains nitrogen.
" " " " . . . . . 233 " carbon.
" " fat " . . . . . 345·6 " "
" " carbohydrate (except lactin) contains 194·2 " "

2. When the composition of a dietary or article of food is stated in terms of grains nitrogen and carbon and ounces fat—

Grains nitrogen × 0·0145 = ounces albuminates.
" " × 3·877 = grains carbon of albuminates.
Oz. fat × 345·6 = " " fat.

and total grains carbon of the dietary or article of food minus the grains carbon of the albuminates and fat × 0·00515 = oz. carbohydrates.

3. When the composition of an article of food is stated in terms of percentage of albuminates, carbohydrates and fats—

Albuminates × 0·158 = nitrogen.
" × 0·532 = carbon of albuminates.
Carbohydrates × 0·444 = " carbohydrates.
Fats × 0·790 = " fats.

XI.—TABLE showing approximately the NUTRITIVE VALUE of various ARTICLES of FOOD. One ounce of each article contains :—

—	Fat per ounce in ounces.	Grains nitrogen per ounce.	Grains carbon per ounce.
Pulses average	0·017	17·86	170
Wheat	0·020	9·22	170
Oatmeal	0·060	8·70	170
Bajri or cumboo	0 026	7·00	170
Maize	0·080	6·93	170
Jowari or cholum	0·026	5·97	170
Barley	0·024	5·81	170
Rice	0·003	5·07	170
Millets, various	0·026	6·40 to 4·09	170
Uncooked meat without bone—lean	0·036	14·22	64
" " " —fat	0·298	9·60	98
Bread, wheaten, from	0·016	8·44	140
" " to		5·55	119
Fish (mackerel)	0·029	16·40	80
" (eels)	0·138	8·75	
Milk (cow's)	0·039	2·75	31
Cheese	0·300	23·00	162
Potatoes	0 002	1·00	49
Carrots	0·005	0 40	18
Biscuit	0·013	22·70	183
Sugar	..	..	187
Fat	1·00	..	345·6
Eggs	0·105	8·07	64

XII.—TABLE of FOOD EQUIVALENTS, showing the QUANTITY (in ounces) of each ARTICLE of FOOD required to yield: A. 100 GRAINS of NITROGEN; B. 2000 GRAINS of CARBON.

—	Ounces yielding 100 grains nitrogen.	Ounces yielding 2000 grains carbon.
Pulses	5·6	
Wheat	10·9	
Oatmeal	11·5	
Bajri or cumboo	14·2	
Maize	14·4	12
Jowari or cholum	16·8	
Barley	17·3	
Rice	20·0	
Millets, various	15·6 to 22	
Uncooked meat without bone, lean	7·0	31·2
Uncooked meat without bone, fat	10·4	20·3
Bread, from	11·8	14·3
Bread, to	18·0	17·0
Fish (white), from	6·1	25·0
Fish (white), to	11 4	
Milk (cow's)	36·4	65·0
Cheese	4·4	12·3
Potatoes	100·0	40·8
Carrots	250·0	111·0
Biscuit	4·4	10·9
Sugar	..	10·7
Fat	..	5·8
Eggs	12·4	31·1

XIII.

The following rules are in force in the Presidency of Bombay (see G. R. Genl. Department, No. 813, of 19th March, 1880).

Rules for the Guidance of Medical Officers forwarding Articles for Analysis in Cases of Suspected Poisoning.

1. In all cases of death from presumed poisoning, the stomach should be tied at both ends and removed from the body, so that its contents may be retained; after removal it should be opened, the contents received into a perfectly clean bottle, and the mucous surface of the stomach carefully examined, its appearance noted, and any suspicious particles found adherent thereto should be picked off with a pair of forceps and placed in a separate small phial for transmission.

2. In all cases of death from presumed poisoning, the mucous membrane of the mouth, pharynx, and œsophagus, should be examined, and any unusual appearance, or marks of corrosion thereon, carefully noted.

3. The following articles should be forwarded for analysis, each in a separate bottle, in cases of death from presumed poisoning:—

A. The stomach.

B. The contents of the stomach.

C. Suspicious particles (if any have been found) removed from the mucous membrane of the stomach.

D. A portion of the liver, not less than sixteen ounces in weight, or the whole liver if the organ weighs less than sixteen ounces, and one kidney —these may be sent in the same bottle.

E. The vomited matter, if any; and in cases where it is suspected that death has been caused by the administration of datura or other vegetable poison:—

F. The contents of the small intestines.

4. Strong methylated spirit should in all cases be added, as laid down in the rules for the transmission by post of articles for analysis (see below) to the contents of bottles *A*, *D*, and *F*, and also to the contents of bottles *B* and *E*, unless it is suspected that alcoholic poisoning has been the cause of death. No spirit need be added to the contents of bottle *C*.

5. All bottles should be carefully sealed by the Medical Officer, and closed in such a manner that they cannot be opened without destroying the seal. The seal used should be the same throughout, and a private seal; each bottle should be labelled, and the labels signed by the Medical Officer.

6. After having made a post-mortem examination in a case of suspected poisoning, the medical officer should report the results thereof to the police, and on receipt of an order from a magistrate, or from a superintendent or assistant-superintendent of police, but not before, forward the viscera, &c., of the deceased to the chemical analyser to Government for examination. In cases where no death has occurred, but where it is suspected that poison has been administered, the medical officer should similarly report the case to the police, and on receipt of an order from a magistrate, superintendent, or assistant-superintendent of police, forward the vomited matter—or contents of the stomach removed by the stomach pump—of the affected individual, or other matters requiring analysis, to the chemical analyser to Government.

7. When, on receipt of the necessary order, a medical officer forwards articles to the chemical analyser for examination, he should address at the same time a letter to the chemical analyser advising their despatch. This letter should contain—

a. An impression of the seal used in closing the bottles, and a description thereof.
b. A list of the articles forwarded, and a statement as to how the articles have been forwarded.
c. The name of the officer from whom the order has been received to forward the articles, and the number and date of such order.
d. A detailed account of the post-mortem appearances observed.
e. If he has seen the case during life, an account of the symptoms observed, and a statement of the treatment, if any, adopted.

8. When articles are forwarded by post to the chemical analyser to Government, each package should be franked externally with the name and address of the medical officer forwarding the articles, and the rules for the transmission of such articles by post (see below) should be attended to.

Rules for the Transmission by Post of Articles for Analyses.

(1.) The suspected viscus, or other portion of the body to be sent for examination, should be enclosed in a glass bottle or jar, fitted with a stopper or sound cork.

(2.) If liable to decomposition, it should be immersed in methylated spirits of wine, which should be used in the proportion of one-third of the bulk of the material.

N.B.—The use of spirits of wine in packing viscera should be invariable, whether the season is hot or cold, and care should be taken that common bázár spirit is not used.

(3.) The stopper or cork should be carefully tied down with bladder or leather, and sealed. To ascertain that it has been securely closed, the bottle or jar should be placed for some minutes with its mouth down.

(4.) The glass bottle or jar should then be placed in a strong wooden or tin box, which should be large enough to allow of a layer of raw cotton, at least three-fourths of an inch thick, being put between the bottle or the jar and the box.

(5.) The box itself should be encased in common gárah cloth, which should be sealed in accordance with the usual rules of the Post Office as to parcels.

(6.) Despatching officers will be held personally responsible that these instructions are carefully followed. Whenever practicable, such parcels should be packed under the immediate supervision of the district civil surgeon.

(7.) At all stations where there is a district civil surgeon the parcels should invariably be sent to the Post Office by that officer, and not by a subordinate officer; but where there is no civil surgeon they may be sent through the sub-divisional officer.

(8.) A declaration of contents to the officials of the postal department is unnecessary, and should not be made.

1. Magistrates, superintendents, and assistant superintendents of police, on instructing medical officers to forward articles for analyses to the chemical analyser to Government, should at the same time address the latter officer, quoting the number and date of their order to the medical officer, and should furnish the chemical analyser with a brief summary of the history of the case.

2. The principal points on which magistrates, &c., in cases of suspected poisoning, should furnish information to the chemical analyser, are as follows:—

a. What interval was there between the last eating or drinking and the first appearance of symptoms of poisoning.
b. What interval was there between the last eating or drinking and death (if this occurred).
c. What were the first symptoms.
d. Were any of the following symptoms present? If so, state which:—
  a. Vomiting and purging.
  b. Deep sleep.
  c. Tingling of the skin and throat.
  d. Convulsions or twitchings of the muscles.
  e. Delirium and clutching at imaginary objects.
e. Were any other symptoms noticed?
f. Did any other persons partake of the suspected food or drink, and did they also suffer from similar or other symptoms of poisoning?

3. Any other information available, likely to prove serviceable as a guide to the class of poison administered, should at the same time be furnished.

4. Any articles in the possession of the police, of which an analysis may be required, may at the same time be forwarded to the chemical analyser, strong methylated spirits of wine being added to such as are likely to undergo rapid decomposition.

5. In forwarding such articles the precautions laid down in Rules 5, 7 *a* and *b*, and 8, for the guidance of medical officers, should be attended to.

XIV.—Statistics of Poisoning in England.

(Blyth on Poisons, p. 30.)

—	Deaths per Ann. Average of Five Years ending 1880.	Ratio per Million of Population.
Homicide	2·4	·07
Suicide	128·4	3·55
Accident	185·4	5·15
Total . . .	316·2	8·77

DEATHS per 100 DEATHS from POISON.

(From Returns of five years ending 1867.)

Opiates	41·4
Lead salts	15·9
Cyanides	11·3
Arsenic	5·5
Oxalic acid	4·3
Mineral acids	4·2
Mercury	3·3
Strychnia	2·7
Alcohol	2·3
Phosphorus	0·9
Other poisons	8·2
Total	100·0

XV.—STATISTICS of POISONING in INDIA.

1. Average number of deaths per annum from poison in the Presidency of Bombay (from Sanitary Commissioner's Reports for the five years ending 1884):—

—	Number.	Ratio per Million of Population.
Homicide (or otherwise)[1]	19·2	1·16
Suicide	62·6	3·79
Accident[2]	30·4	1·85
Total	112·2	6·80

[1] *i.e.*, still under judicial investigation.
[2] This does not include 1192 deaths from snake-bite, equal to 72·24 per 1,000,000 of population

2. Deaths from poison shown in the Chemical Examiner's Reports for the year 1884:—

—	Arsenic.	Opium.	Datura.	Other.	Total
Bengal	22	53	..	8	83
Madras	19	4	..	8	31
Bombay	22	9	1	3	35
Punjaub	45	18	1	1	65
N W. Provinces	29	17	1	1	48
Total	137	101	3	21	262
Per cent. of deaths from poison	52·3	38·5	1·2	8·0	100·0

3. Bombay Presidency: poisons detected during the ten years ending 1884 (from Chemical Analyser's Reports):—

	Cases
Arsenic[1]	507
Opium	151
Datura	74
Copper	50
Mercury	29
Lead	19
Other metals	5
Pounded glass	31
Strychnia	17
Oleanders	8
Aconite	6
Plumbago	6
Alcohol	6
Cucurbitaceous tubers	5
Oxalic acid	4
Acid and alkaline corrosives	3
Cyanides	3
Other poisons	23
Total	947

[1] In 450 arsenious oxide only, in 13 mixed arsenious oxide and sulphides, in 3½ sulphides only, and in 10 arsenites of copper.

4. Bombay Presidency: persons poisoned and deaths from poison during the ten years ending 1884 (from Chemical Analyser's Reports):—

—	Persons Poisoned.	Deaths from Poison.
Arsenic	652	305
Opium	142	131
Datura	146	22
Other poisons	155	42[2]
Total	1095	500

[2] Eight of these from strychnia and four from oxalic acid.

5. Cattle poisoning: deaths of cattle from poison in India in 1884 (from Reports of Chemical Examiners):—

Bengal	41
Madras	38
Bombay	51
Punjaub	147
N.W. Provinces	11
Total	292[3]

[3] All except four from arsenical poisoning.

6. Cattle poisoning in Bombay Presidency: poisons detected in the ten years ending 1884:—

Arsenious oxide	696[1]
" " with sulphides	9
" " with oxides of lead	9
Sulphides of arsenic only	11
Oxides of lead only	6
Sulphate of copper	6
Nux vomica	1
Cocculus indicus	3
Mercury	1
Sulphate of iron	1
Total	743[2]

[1] Mixed in three cases with pounded glass, in two with compounds of mercury, and in one with sulphate of copper.

[2] Altogether 677 animals died from poison in the ten years.

XVI.

Inspecting Veterinary Surgeon W. Lamb, in a letter to the Government of Bombay, dated 5th December, 1872, gives the following summary of the symptoms likely to be observed in different cases of cattle poisoning:—

1st. Poisoning with arsenic, or other irritant mineral poisons.

The animal evinces soon, or almost immediately after the poison is given, violent abdominal pain, trembles, writhes, rolls about in agony, groans, foams at the mouth, and dies speedily. In more protracted cases, from less poison being given, or from its being absorbed more slowly, the abdomen swells, acute diarrhœa or dysentery supervenes, great thirst prevails, and death ensues from exhaustion.

Rinderpest, hoven, and dysentery are the diseases whose symptoms most nearly resemble those of arsenical poisoning. The following are the most obvious distinguishing marks:—

(*a'*) In rinderpest there is trembling with nervous or spasmodic twitches, but accompanied at first by dulness instead of agony. The access of the disease is by comparison gradual, for irritant poison produces violent symptoms suddenly.

(*b'*) In hoven distension of the abdomen precedes and causes pain: in poisoning by arsenic, pain precedes distension; it is also more acute, violent, and agonizing.

(*c'*). In acute dysentery, violent bloody slimy purging is the prominent symptom; abdominal pain is subsidiary. In arsenical poisoning pain is the chief and primary symptom; purging may come after.

2nd. Aconite.—I have not myself seen cattle suffering from this poison, nor am I aware that the symptoms thereof have anywhere been recorded; but they may be expected to be the same as, or similar to, those that occur in horses under such circumstances. Of these, the most characteristic are trembling, nausea, eructations, retchings causing discharge of mucus, partial convulsions, early inability to stand, paralysis, stupor, pulse and breathing slow, coma, death.

3rd. Nux vomica, in poisonous doses, causes spasms of the voluntary muscles, and all the symptoms of acute tetanus, such as jaws fixed, the haws projected over the eyes, ears erect, body rigid and often contorted, tail extended and quivering, death being caused by suffocation from the diaphragm and other muscles of respiration being affected, or by the beating of the heart being stopped by spasmodic contraction.

Tetanus might be mistaken for this form of poisoning: but it is a rare disease

among horned cattle, and is hardly ever caused in them except by wounds, and is never so sudden and violent in its attack, nor so rapidly fatal, as the poison of nux vomica.

The following rules for the guidance of officers forwarding articles for analysis in cases of suspected cattle poisoning, were published by the Government of Bombay on the 24th December, 1872:—

"The whole of the contents of the stomach and upper portion of the intestines should, in the first place, be emptied out into a suitable clean vessel; a portion of the same, say about as much as would fill a 12-oz. bottle, should then be removed and placed in a clean glass bottle, and the remainder, if any, washed out with water, and carefully searched for any mineral masses, fragments of roots, suspicious-looking seeds, &c.; any such substances being discovered, should be placed in a separate paper packet.

"2. A portion of the stomach, a portion of the liver, and a portion of one kidney, enough, when taken, to fill say a 20-oz. wide-mouthed bottle, should then be cut off, and placed together in one such bottle, along with some spirits of wine, or strong country spirit.

"3. Both bottles, viz., that containing the portion of the contents of the stomach, and the other containing the portions of the solid organs, as mentioned above, are to be labelled, corked, and sealed with a private seal. The packet containing the mineral masses, portions of root, &c., is also to be labelled and sealed with the same seal; and the two bottles and packet are then to be made up into a parcel, and forwarded to the Chemical Analyser to Government, Bombay.

"4. The letter advising the despatch of the parcel should contain a full account of the case, giving the symptoms observed, any unusual post-mortem appearances noted, &c., &c. Attached to the letter should be an impression of the private seal used in securing the bottles and packet."

XVII.—Note on the Question of Access in Legitimacy Cases.

The English law on this subject is to be found in the answers given by the judges to questions put to them by the House of Lords in the *Banbury Peerage Case*, 1 S. & S. 155, A.D. 1811. The law, as then stated, was recognised in a subsequent case, in the House of Lords, in 1837 (*Morris* v. *Davis*, 5 Cl. & F., p. 163), and is as follows:—1st. That when the husband and wife have opportunities of access, the presumption of legitimacy may be rebutted by circumstances inducing a contrary presumption. 2nd. That non-access or non-generating access may be proved by means of such legal evidence as is admissible in every other case in which it is necessary to prove a physical fact. 3rd. That after proof of sexual intercourse evidence will not be admitted, except to disprove the fact. 4th. That sexual intercourse is presumed, unless met by such evidence as satisfies those who are to decide that it did not take place.

By "access" is meant sexual intercourse, and not such intercourse as is understood by being in the same place or in the same house (*Banbury Peerage Case*; *Morris* v. *Davis*). Although possibility of such access may be proved, yet, if the Court is satisfied, from legal evidence, that no sexual intercourse did take place, the presumption of legitimacy is rebutted. In the case of *Aylesford* v. *Aylesford*, reported in the *Times* of July 3rd, 1885, the husband and wife were both living in London during the period, or some portions of the period, when the child whose legitimacy was in question could have been begotten. The circumstances of the case negatived the probability of intercourse between the husband and wife, although it was possible. The child was found, by the House of Lords, to be illegitimate. In the case of *In re Westhead's Trusts* (*Times*, July 29th, 1885), there was no evidence where the husband was during the critical period during which the child could have been begotten. He, however, had at that time taken divorce proceedings against his wife. The Court of Appeal held that the circum-

stances of the case negatived any probability of intercourse between the husband and wife, and, being satisfied it had not taken place, held the child to be illegitimate.

In the case of *Rex* v. *Inhabitants of Mansfield*, 1 Q. B. 444, the Court of Queen's Bench determined that the non-access of the husband might be proved by circumstances, "one of which," it was said, "certainly is adulterous intercourse between the husband or wife and another party." In that case the whole proof consisted only of that single fact, and it was held not sufficient to rebut the presumption. The parties, however, were in a low class of life, the wife being a pauper, circumstances which Kay, J., in *Hawes* v. *Draegen*, 23 Ch. Div., p. 173, said must be taken into consideration in determining whether the presumption is rebutted.

The Courts in India would no doubt construe the 112th sect. of the Evidence Act in accordance with the English decisions. It will be noticed that the 112th sect. does not in terms refer to the presumption being rebutted if the husband be impotent, but proof of such impotency would negative the fact of "access" in the sense in which it is submitted the word is used in the above section.

XVIII.—Characters of the Embryo at various Periods anterior to the End of the Fourth Month (Guy).

Embryo of three to four weeks.—Length, about ⅓ inch. Weight, about twenty grains. Size, that of a large ant or barleycorn. Form, that of a serpent, the head indicated by a swelling, the caudal extremity slender, and ending in the umbilical cord; the mouth indicated by a cleft, the eyes by two black points, the members appearing as nipple-like protuberances. The villosities of the chorion uniformly spread over the surface.

Embryo of six weeks.—Length, from ½ inch to less than 1 inch. Weight, from forty to seventy-five grains. The head distinct from the chest, and the face from the cranium; apertures of the nose, mouth, eyes, and ears perceptible; the hands and forearms in the middle of the length, and the fingers distinct; the legs and feet situated near the anus; a distinct umbilicus for the attachment of the cord, which consists of the omphalo-mesenteric vessels, of part of the urachus, of the intestinal tube, and of filaments which represent the umbilical vessels. The placenta forming; the chorion and amnion still separate; the umbilical vesicle very large. Points of ossification in the clavicle and maxillary bone.

Embryo of two months.—Length variously stated at from 1½ to 4 inches (Tidy gives the length as 16 to 19 lines). Weight, 120 to 300 grains. Rudiments of nose, lips, and eyelids; organs of generation visible; arms and legs detached from the trunk, anus marked by a dark spot. Rudiments of lungs, spleen, and supra-renal capsules, cæcum behind the umbilicus, digestive canal withdrawn into the abdomen; urachus visible; chorion beginning to touch the amnion, at the point opposite to the insertion of the placenta, which begins to assume its regular form; umbilical vessels becoming twisted.—Points of ossification in frontal bone and ribs; also in the shafts of the long bones of the extremities, in the shafts of the metacarpal and metatarsal bones and phalanges, and in the scapula and ilium (Tidy).

Embryo of three months.—Length variously stated at 2 to 6 inches (2 to 2½ inches, Tidy). Weight, one to three ounces (480 to 720 grains, Tidy). The head voluminous; the eyelids and lips in contact; membrana pupillaris visible; fingers separated; lower extremities longer than rudimentary tail (clitoris or penis very long. Tidy). Thymus and supra-renal capsules present; the ventricle of the heart distinct. The decidua uterina and reflexa in contact. The funis containing umbilical vessels and a little gelatinous matter; placenta completely isolated; the umbilical vesicle, allantois, and omphalo-mesenteric vessels have disappeared (points of ossification for the occipital, sphenoid, os unguis, nasal bones, squamous portion of temporal and ischium. Tidy).

XIX.—(See also pp. 346 *et seq.*)

EXTERNAL CHARACTERS of the FŒTUS at the END of EACH MONTH of UTERINE LIFE.

Month	4	5	6	7	8	9
Average length in inches Mean weight (Guy)	6½ 5 oz.	8½ 11 oz.	14 2 lbs. 2 oz.	15 3 lbs. 8 oz.	17 4 lbs. 5 oz.	19½ 6 lbs. 8 oz.
Skin	No sebaceous covering or fibrous structure apparent.		Fibrous structure, papillæ and sebaceous matter beginning to appear, covered with down.	Dusky red; thick and fibrous, and covered with sebaceous matter.	Covered with fine short hairs and sebaceous matter.	Down almost all disappeared; covered with sebaceous matter
Nails	Appearing.	Very distinct.	Growing.	Do not quite reach to end of fingers.	Reach to end of fingers.	
Hair on scalp	None.	Appearing.	Distinct.	About a quarter of an inch long.	Over a quarter of an inch long.	About one inch long.
Eyes, &c.	Lids adherent; membrana pupillaris distinct.		Lids adherent; membrana pupillaris distinct; eyebrows and eyelashes beginning.	Lids non-adherent; membrana pupillaris getting indistinct.	Membrana pupillaris hardly visible.	
Position of middle point of body.	On sternum.	On sternum.	At lower end of sternum.	Below lower end of sternum.	Nearer umbilicus than sternum.	Just above the umbilicus.

XX.—(See also pp. 346 *et seq.*)

INTERNAL CHARACTERS of FŒTUS at END of EACH MONTH of UTERINE LIFE.

Month	4	5	6	7	8	9
Ossification apparent in	Ossicles of ear* and upper parts of sacrum.	Pubis and os calcis.	Four divisions of sternum.	Astragalus.	Last sacral vertebra.	Lower epiphysis of femur.
Intestines	Cæcum near right kidney, valve visible.	..	Sacculi appearing in large intestine.	Cæcum in right iliac fossa, valvulæ conniventes appearing.		
Meconium	In duodenum.	At commencement of large intestine.	In upper part of large intestine.	Present nearly throughout large intestine.	..	Has reached the rectum.
Gall bladder . . .	Appearing.	Distinct.	Contains serous fluid.	Contains bile.		
Position of testicles .	..	..	Close to kidneys.	Between kidneys and internal ring.	At or in the internal ring.	Generally external.
Miscellaneous . .	..	Germs of permanent teeth apparent.	Cerebral hemispheres cover cerebellum.	Cerebral convolutions becoming apparent.		

* For points of ossification appearing before the 4th month, see Table of Characters of Embryo anterior to the 4th Month (XVIII.).

XXI.—Guy's Table (based on Billard's Observations) showing the Proportion of Cases in which the Foramen Ovall and Ductus Venosus have been found open at various Periods after Birth.

The third column shows (calculated from Tardieu as quoted by Tidy) the proportion of cases in which separation of the cord was found to have taken place.

Day	Foramen Ovale open in Cases, per cent	Ductus Arteriosus open in Cases, per cent	Cord separated in Cases, per cent.
1	74	68	..
2	68	59	3
3	64	68	17
4	63	63	40
5	45	52	70
6	..	..	90
7	..	..	95
8	25	15	97

XXII.—Tidy's Table of the Changes which occur during the First few Days after Birth.

Period after birth.	Conditions observed.
A few minutes to some hours	The stomach contains a frothy fluid and clots will be found in the vessels of the umbilical cord.
After 24 hours . . .	Concentric thickening of the umbilical arteries near umbilicus.
After 2nd day . . .	Contraction throughout the greater part of the umbilical arteries. Epidermis beginning to exfoliate.
After 3rd day . . .	Umbilical arteries contracted throughout. Slight contraction of the umbilical veins. Formation of inflamed ring round cord.
After 4th day . . .	Cord separated.
After 5th day . . .	Contraction of umbilical veins complete.
After 7th day . . .	Ductus arteriosus contracted to size of a crow-quill.
8th to 10th day . . .	Fœtal circulatory openings obliterated.
10th to 12th day. . .	Osseous centre of lower femoral epiphysis larger in diameter than ¼th to ⅓th of an inch. Cicatrization of umbilicus. (If the umbilicus be healed, it indicates life for about 21 days.)

XXIII.—24 & 25 Vict. Cap. 100, Sects. 58 & 59.

58. "Every woman being with child who, with intent to procure her own miscarriage, shall unlawfully administer to herself any poison or other noxious thing, or shall unlawfully use any instrument or other means whatsoever with like intent, and whosoever, with intent to procure the miscarriage of any woman, whether she be or be not with child, shall unlawfully administer, &c., shall be guilty of felony."

59. "Whosoever shall unlawfully supply or procure any poison or other noxious thing, or any instrument or thing whatsoever, knowing that the same is intended to be unlawfully used or employed with intent to procure the miscarriage of any woman, whether she be or be not with child, shall be guilty of misdemeanour, and being convicted thereof, shall be liable, at the discretion of the Court, to be kept in penal servitude for the term of three years, or to be imprisoned for any term not exceeding two years."

N.B.—By 27 & 28 Vict. c. 47, s. 2, the minimum term of penal servitude awardable is increased to five years.

XXIV.—Abstract of Tables of Expectation of Life in England.

(From Woodman and Tidy, p. 608.)

Age.	Farr's Table of Expectation among the Population generally.			Expectation of Healthy Lives from the experience of twenty Life Offices published in 1869.			Age.
	Males.	Females.	Both Sexes.	Males.	Females.	Both Sexes.	
20	40	40	41	42·09	40·87	41·98	20
21	40	39	40	41·33	40·17	41·23	21
22	39	39	39	40·62	39·40	40·51	22
23	38	38	39	39·87	38·61	39·84	23
24	38	37	38	39·18	37·95	39·15	24
25	37	36	38	38·44	37·40	38·44	25
26	36	36	37	37·64	36·80	37·65	26
27	36	35	36	36·90	36·21	36·93	27
28	35	34	36	36·14	35·69	36·18	28
29	34	34	35	35·12	35·07	35·47	29
30	34	33	34	34·68	34·55	34·75	30
31	33	32	34	33·96	33·90	34·04	31
32	32	32	33	33·21	33·27	33·30	32
33	32	31	32	32·48	32·75	32·59	33
34	31	30	32	31·75	32·04	31·86	34
35	30	30	31	31·03	31·43	31·15	35
36	30	29	30	30·28	30·80	30·41	36
37	29	29	30	29·55	30·21	29·69	37
38	28	28	29	28·83	29·55	28·97	38
39	28	27	28	28·12	28·88	28·27	39
40	27	27	28	27·42	28·24	27·57	40
41	26	26	27	26·69	27·59	26·85	41
42	25	25	26	25·96	26·97	26·14	42
43	25	25	26	25·24	26·30	25·42	43
44	24	24	25	24·50	25·68	24·69	44
45	24	23	24	23·79	24·96	23·98	45
46	23	23	24	23·08	24·26	23·27	46
47	23	22	23	22·37	23 68	22·57	47
48	22	21	22	21·68	23·02	21·89	48
49	21	21	22	20·98	22·34	21·20	49
50	21	20	21	20·30	21·60	20·51	50
51	20	19	20	19·63	20·94	19·84	51
52	19	19	20	18·97	20·18	19·17	52
53	19	18	19	18·29	19·60	18·50	53
54	18	17	18	17·60	18·86	17·81	54
55	17	17	18	16·93	18·17	17·14	55
56	17	16	17	16·32	17·53	16·53	56
57	16	15	16	15·69	16·88	15·90	57
58	15	15	16	15·07	16·16	15·26	58
59	15	14	15	14·44	15·56	15·64	59
60	14	14	14	13·80	14·85	13·99	60
61	13	13	14	13·23	14·21	13·42	61
62	13	12	13	12·66	13·50	12·83	62
63	12	12	13	12·09	12·92	12·26	63
64	12	11	12	11·54	12·38	11·72	64
65	11	11	12	11·01	11·78	11·17	65
66	11	10	11	10·49	11·24	10·65	66
67	10	10	10	9·98	10·63	10·12	67
68	10	9	10	9·46	10·10	9·61	68
69	9	9	10	8·99	9·60	9·13	69
70	9	9	9	8·54	9·12	8·68	70

XXV.—Table of "Pure, or Unloaded, 3 per cent. Premiums," Corresponding to Certain Expectations.

The "pure or unloaded 3 per cent. premium" is the sum which, if paid yearly during the period of expectation, will at 3 per cent. interest amount at the end of that period to the sum assured. Assurance offices, of course, add to the "unloaded premium" a certain percentage termed the "loading," to cover cost of management and profit.

Age.	Expectation of Healthy Male Lives from experience of twenty Life Offices.	Unloaded 3 per cent. premium in sterling per £100 assured.	Unloaded 3 per cent. premium stated as percentage on the sum assured.
		£ s. d	
20	42·06	1 8 7	1·430
25	38·44	1 12 6	1·625
30	34·68	1 17 7	1·880
35	31·03	2 3 10	2·190
40	27·40	2 11 9	2·588
45	23·79	3 2 3	3·112
50	20·31	3 16 0	3·800
55	16·93	4 14 6	4·725
60	13·83	5 19 9	5·988
65	11·01	7 14 1	7·705

XXVI.—Abstracts of Tables of Expectation of Life in India, viz.,

A. Of a Table of expectation of life compiled by J. Westland, Esq., Beng C S., from the experience of the Bengal Uncovenanted Civil Service Family Pension Fund; European and Eurasian lives; period of observation, 1837 to 1862.

B. Of a Table of expectation of life compiled by A. F. Cox, Esq., Mad. C.S., from the combined mortality statistics of the Bengal, Madras, and Bombay Civil Services; European lives only; periods of observation—Bengal, 1850 to 1872; Madras, 1790 to 1852; Bombay, 1790 to 1860; number of lives under observation—Bengal, 600 to 1200; Madras, 500; Bombay, 700.

Note.—Table A is based wholly on Indian experience; Table B on Indian experience only up to the age of fifty.

Age.	Expectation A.	Expectation B	Age.	Expectation A.	Expectation B.	Age.	Expectation A.	Expectation B
20	..	33·65	42	17·86	22·12	62	7·93	11·39
22	31·43	32 49	44	16·69	21·18	64	7·27	10·54
24	29·85	31·41	46	15·57	20·29	66	6·62	9 52
26	28 32	30·36	48	14·53	19·39	68	5·92	8·54
28	26·84	29·31	50	13·55	18·43	70	5·20	7·62
30	25·39	28·26	52	12·63	17·38	72	4·50	6 75
32	24 02	27·22	54	11·71	16·25	74	3·80	5·95
34	22·72	26·19	56	10·72	15·09	76	3·10	5·23
36	21·49	25·16	58	9·68	13 91	78	2·41	4·57
38	20·28	24·13	60	8·72	12 74	80	1·72	3·98
40	19·07	23·10						

XXVII.—MALE EUROPEANS. CORRESPONDENCE BETWEEN HEIGHT and WEIGHT, CIRCUMFERENCE of CHEST and VITAL CAPACITY.

Height in feet and inches.	Model weights at age thirty (Liharzik) in lbs.	Weights (Allen) in lbs.	Circumference of Chest in inches (Allen).	Vital Capacity in Cubic inches (Hutchinson).
Ft. Ins.	Lbs.	Lbs.	Ins.	Cubic Ins.
5 1	116	120	34·06	175
5 2	126	125	35·13	177
5 3	133	130	35·70	189
5 4	139	135	36·26	193
5 5	142	140	36·83	201
5 6	145	143	37·50	214
5 7	148	145	38·16	229
5 8	155	148	38·53	228
5 9	162	155	39·10	237
5 10	169	160	39·66	246
5 11	174	165	40·23	247
6 0	178	170	40·80	259

	Ft.	Ins.
Average height of		
Englishmen brought up under favourable circumstances .	5	9¾
Englishwomen " " " .	5	2 (Sieveking)
Belgians, Males	5	6
" Females (Quetelet)	5	2

Mean vital capacity, men of all heights (Hutchinson) . . 217 cubic inches.

Vital capacity attains its maximum at the age of 35 years.

XXVIII.—SCHEDULE, ACT No. 36 of 1858.

FORM A.

CERTIFICATE OF MEDICAL OFFICER.—(*See Sections IV. and VIII.*)

I, the undersigned (*here enter name and official designation*), hereby certify that I, on the day of at , personally examined (*here enter name and residence of Lunatic*), and that the said is a lunatic (*or an idiot, or a person of unsound mind*) and a proper person to be taken charge of, and detained under care and treatment, and that I have formed this opinion on the following grounds, namely:—

1. Facts indicating insanity observed by myself (*here state the facts*).
2. Other facts (if any) indicating insanity communicated to me by others (*here state the information and from whom*).

(Signed) _____

FORM B.

ORDER FOR THE RECEPTION OF A PRIVATE PATIENT.—(*See Section VII.*)

I, the undersigned, hereby request you to receive A. B., a Lunatic (*or an idiot, or a person of unsound mind*), as a patient into your Asylum. Subjoined is a statement respecting the said A. B.

(Signed) name
Occupation (if any)
Place of abode
Degree of relationship (if any), or other circumstance of connection with the patient.

Dated this day of one thousand eight hundred and

To Superintendent of the Asylum at (*describing the Asylum*).

STATEMENT.

[*If any of the particulars in this Statement be not known, the fact to be so stated*].

Name of patient, with Christian name at length.
Sex and age.
Married, single, or widowed.
Condition of life, and previous occupation (if any).
The religious persuasion, as far as known.
Previous place of abode.
Whether first attack.
Age (if known) on first attack.
When and where previously under care and treatment.
Duration of existing attack.
Supposed cause.
Whether subject to epilepsy.
Whether suicidal.
Whether dangerous to others.
Whether found lunatic by inquisition or inquiry under order of Court, and date of Commission or order for inquisition or enquiry.
Whether any member of patient's family has been or is affected with insanity.

(Signed) name.

[*Where the person signing the Statement is not the person who signs the order the following particulars concerning the person signing the Statement are to be added; namely,*]

Occupation (if any).
Place of abode.
Degree of relationship (if any), or other circumstances of connection with the patient.

XXIX.—Internal Examination of the Body.

General Directions.—(1) The order of examination should be that here given, unless special reasons of the nature indicated at page 38 exist for departure therefrom. (2) Incisions made through the skin for the purpose of opening cavities should avoid already existing external wounds. (3) If on dissection any internal injury is found likely to have resulted from external violence, careful examination should be made—if this has not already been done—for signs of violence in the tissues between the seat of injury and the surface of the body; and for marks of violence on the surface of the body over the seat of injury. (4) Any unusual appearances found, not specially mentioned under appearances to be looked for, should always be recorded.

I.—The Head.

The internal examination of the body should commence with dissection of this cavity:—(*a*) in cases where the cause of death is doubtful, and (*b*) when it is suspected that death has been due to head injury, or has occurred by coma.

Procedure.	Appearances to be looked for and recorded.
1. Make an incision through the integuments from ear to ear over the vertex and reflect the scalp, one flap forwards, the other backwards.	(*a*) Extravasations of blood in or under the scalp, their situation and extent. (*b*) Injuries to the bones of the skull-cap visible externally, viz., separation of sutures, fractures, or indentations; their situation, extent, and direction (see 2 *b*, below).
2. Saw through the skull by a circular cut at the level of about an inch above the orbits in front, and of the occipital protuberance behind, detaching the skull cap without using the chisel and mallet (unless it is certain that there is no fracture of the skull). Raise the skull cap from before backwards, separating the dura mater from it. 3. Divide and reflect the dura mater on either side. N.B.—If the dura mater is so firmly adherent to the skull cap as not to be easily separable, it should be divided carefully and removed with the skull cap.	(*a*) Unusual thinness of the skull bones. (*b*) Complete 1 *b*, above, by examining inner surface of skull-cap. (*c*) Fulness (or the reverse) of the longitudinal sinus. (*d*) Condition of the membranes of the brain, *e.g.*, amount of adhesion, if any, of the dura mater to the skull-cap, presence of congestion or signs of disease. (*e*) Extravasations of blood between the skull-cap and the brain, their situation and extent.
4. Remove the brain carefully, place it base downwards, and proceed to slice it horizontally from above.	(*a*) Extravasations at the base of the skull, their situation and extent. (*b*) Volume of any serous fluid found within the skull or ventricles of the brain. (*c*) Weight, colour, and consistence of the brain (and in immature infants its condition of development, see p. 349). (*d*) Apoplectic effusions within the substance of the brain, their situation and extent. (*e*) Freedom or otherwise from disease of the coats of the cerebral arteries.
5. Strip off the dura mater from the interior of the skull.	(*a*) Fractures of the base or lateral portions of the skull, their situation, extent, and direction, and thickness of the bones at the seat of the fracture. (*b*) In infants, presence of air in the cavity of the tympanum (see p. 361).
6. Examine the upper portion of the spinal cord through the foramen magnum.	If any signs of injury to the cord or upper cervical vertebræ are found, proceed at once to IV., returning subsequently to II.

II.—The Thorax (including preliminary examination of the abdominal cavity).

The internal examination of the body should commence here, in cases where death appears to have been due to chest injury, or to have occurred by asphyxia. Also when there is reason to believe that the cause of death is connected with the contents of the abdomen. In this last case after II. 1, proceed to III. (see N.B. below).

Procedure.	Appearances to be looked for and recorded.
1. Make a long incision from a little above the sternum down to the pubes, reflect the integuments on either side, laying open the abdominal cavity but not the cavity of the thorax. In infants take care to carry the incision a little to the left of the umbilicus.	(*a*) Position, colour, and general appearance of the exposed viscera. (*b*) Presence of abnormal contents, *e.g.*: blood, products of inflammation, or tumours. (*c*) Determine with the hand (especially in new-born infants) the position of the diaphragm, noting whether the upper level of this is between the fifth and sixth ribs, or higher. N.B.—Should this preliminary examination indicate, or there be reason to believe, that the cause of death is connected with the contents of the abdomen, proceed at once to III., subsequently returning to II. 2.
2. Complete the reflection of the integuments over the thorax to a point beyond the junction of the cartilages of the ribs.	(*a*) Extravasations of blood or signs of bruising in the integuments of the front of the chest, their situation, and extent. (*b*) Fractures of the anterior portions of the ribs, their situation, and the direction in which the fractured ends appear to have been driven (see also II. 9 *c*).
3. Divide the rib cartilages as far from the sternum as possible, or, if these are ossified, cut through the ribs a little outside the cartilages. Cutting upwards, close under the rib cartilages, raise the sternum and cartilages, and very carefully cut through the sterno-clavicular joints, taking care not to wound the underlying large vessels. Remove the sternum and attached rib cartilages.	(*a*) Volume of the lungs, *i.e.*: whether projecting out of the chest (indicating emphysema); or expanded and nearly covering the pericardium, but not projecting; or collapsed, exposing the pericardium. (*b*) Colour of the lungs; in infants, whether dark red or bright red. (*c*) Fluid in the pleural cavities, nature and volume. (*d*) Adhesions of the lungs. (*e*) Condition of the thymus gland. (*f*) Tumours in the thorax.
4. Open the pericardium.	(*a*) Condition of the pericardium; nature and volume of any fluid present therein (see p. 86). (*b*) Size, colour, and consistence of the heart, and condition of fulness of the coronary vessels.

Procedure.	Appearances to be looked for and recorded.
5. Without removing the heart from the body, open its cavities in the following order: (1) R. ventricle; (2) R auricle; (3) L. auricle; (4) L. ventricle. Incisions required: (1) Along the right border of the heart, beginning close to the base and ending short of the apex. (2) Begins midway between the entrances of the venæ cavæ, and ends just in front of the base. (3) Begins at the left superior pulmonary vein, and ends just in front of the base, short of the coronary vein. (4) Begins behind the base and ends short of the apex.	Note, as each cavity is opened, the amount and condition of its contents.
6. Remove the lungs and heart together. N.B.—In cases where it is suspected that death has been due to injury to the neck, and in cases where the condition of or presence of foreign matters in the gullet or air-passages is likely to be of importance, proceed after 5 to 10, returning to 6 *et seq.* afterwards.	(*a*) Presence on the surface of the lungs of Tardieu's spots, or patches of emphysema (see Strangulation and Suffocation, pp. 85 and 89). (*b*) In new-born infants, note if inflated air-vesicles are visible on the surface of the lung, and distinguish between these and bubbles of gas due to putrefaction (see p. 355); then proceed to the hydrostatic test (see p. 356), noting while dividing the lungs whether they exude frothy blood freely and crepitate, or show signs of disease.
7. Separate the heart, and test the condition of the arterial openings by pouring in water.	(*a*) Condition of the cardiac valves, tufts of fibrin on their edges, &c. (*b*) Livid patches on the endocardium (see Arsenic, p. 145). (*c*) Condition of the heart tissue. (*d*) In new-born infants, condition of the foramen ovale. (*e*) Conditions of the portions of the large vessels remaining attached to the heart.
8. Make long incisions into each lung, and, if necessary, follow the branches of the bronchial tubes and pulmonary artery by dividing them with scissors.	(*a*) General characters of the lung tissue. (*b*) Disease of the lungs. (*c*) Apoplectic effusions into the lung substance (see Strangulation, p. 85). (*d*) Condition of the bronchial tubes, nature and quantity of foreign matters present therein (see Drowning, p. 78). (*e*) Condition of the branches of the pulmonary artery, noting any obstruction.
9. Previous to opening the descending aorta, tie two ligatures round the œsophagus near the diaphragm, divide the œsophagus between them and dissect it out of the way.	(*a*) In new-born infants, note the condition of the ductus arteriosus. (*b*) Examine the aorta for atheroma and aneurism. (*c*) Complete the examination of the ribs for fractures (see p. 63).

Procedure.	Appearances to be looked for and recorded.
10. Prolong the incision upwards to the chin, reflect the skin as far back as possible; separate the soft parts from the inside of the lower jaw, cutting close to the bone. Pull the tongue forwards below the chin, and carry the dissection backwards, separating the pharynx and œsophagus with the larynx and trachea from the spine. Open in succession the larynx, trachea and œsophagus.	(*a*) Foreign bodies, marks of corrosion, &c., in the larynx, trachea, and œsophagus. (*b*) Examine the large vessels of the neck for injury, obstruction, &c., opening them carefully. (*c*) Ascertain the condition of the cervical vertebræ.

III.—The Abdomen.

In infants the condition of the umbilical vessels, and of their continuations within the abdomen, should, so far as can be done without removal of any of the abdominal viscera, now be ascertained, completing the examination after the remaining portions become exposed by removal of the viscera.

In all cases examine first generally, and without further dissection, the abdominal viscera *in situ*. Should this examination show, or should there be reason to believe, that the cause of death is connected with any particular organ, the further examination should commence with the organ or organs concerned, *e.g.*, in cases of poisoning the further examination should commence with the stomach and intestines. Each viscus should be carefully examined *in situ* previous to its removal for further examination, noting particularly any enlargement or unusual appearance, and any wound or sign of injury. If a wound be present, its precise situation, direction, and appearance should be described, and it should be noted whether or no any blood is effused in its neighbourhood. The general consistence of the injured viscus should also be noted. Each viscus is then to be removed, and, after removal, further examined as below.

1. **The Liver.**—Note its weight and appearance on section. This may be (*a*) uniform dark brown = *normal*, or (*b*) either uniform dark red, the cut surface exuding blood pretty freely; or of a nutmeg appearance, *i.e.*, in some places dark red, in others buff or yellow = *congestion;* or (*c*) texture dense and tough, surface irregular = *cirrhosis;* or (*d*) in places soft pale yellow, and greasy = *fatty;* or (*e*) uniformly pale yellow, and reduced in size = *yellow atrophy;* or (*f*) enlarged and heavy, consistence doughy, cut surface greyish and glistening or semi-translucent in appearance = *amyloid or lardaceous.* Note presence of abscesses or tumours. Note also the condition of the gall-bladder, and the nature of its contents. If there be any reason to suspect death from poison, preserve for analysis a portion of the liver, at least one pound in weight.

2. **The Spleen.**—Note its size, weight, and appearance on section. If death has been due to rupture of this organ, it is of special importance to note its consistence, and whether or no any signs of injury are present in the surface of the body over it, or in the tissues lying between it and the surface of the body.

3. **The Kidneys.**—Note in regard to each its weight, and whether or no the capsule peels off readily. Then commencing at the convex border make a long incision through it as far as the pelvis, and note if any signs of inflammation of the lining membrane be present. Note the condition of the cut surface. Congestion accompanied by softening and enlargement, or pallor similarly accompanied, indicate inflammation respectively in the early and later stage. Again, the capsule may be adherent, the viscus reduced in size, and its section granular or cystic = *forms of chronic Bright's disease;* or the kidney may be enlarged, the capsule non-adherent, and the section pale, waxy, smooth and glistening = *amyloid degeneration.* Note the presence of morbid growths or tubercular deposit, and in infants note if there is any appearance of blocking of the kidney tubes with uric acid or urates (see page 363). In cases of poisoning preserve one or both kidneys for analysis.

4. **The Pelvic Organs.**—Tie two ligatures round the lower part of the large intestine a little above the rectum, and divide the gut between them. Open the urinary bladder *in situ* and determine its contents, preserving any urine found for subsequent analysis. In male infants note the position of the testicles. Then having examined each organ *in situ*, remove the whole of the pelvic organs together, and complete the examination of the bladder, ureters, and urethra, noting in males the size of the prostate, and the condition of the testicles. Examine the rectum, noting specially in infants the presence or absence of meconium.

In females, examine the generative organs as follows:—

(*a*) **The Vagina.**—This is to be opened first and examined for marks of injury and presence of foreign bodies, preserving any matters found for analysis. Its colour, the presence or absence of rugæ, and the condition of the hymen are also to be noted.

(*b*) **The Uterus.**—Measure externally its length and greatest breadth. Take its weight. Then open it by an incision from fundus to cervix, and note the dimensions of its cavity and the thickness of its walls (see pages 350 and 380). Note the nature of its contents, if any, and if a fœtus be present determine its age (see *Appendix XVIII. to XX.*). Record the condition and colour of the lining membrane and muscular substance, presence of internal injuries, or of morbid growths.

(*c*) **The Ovaries.**—Note in regard to these their size and

external appearance, and after section examine for signs of disease and for true and false corpora lutea (see page 381).

5. **The Stomach.**—Before removing this viscus tie two ligatures round the duodenum close to the stomach, and divide the gut between them. If the abdominal cavity is dissected before the thorax, a similar procedure must, before removing the stomach, be adopted with the œsophagus (see II. 9). In infants, before opening the stomach, note if any air appears to be contained in it (see page 358). Then open the stomach along its lesser curvature, collecting its contents in a clean vessel. Note the volume of the fluid contained in the stomach, its general appearance, the character of any matters suspended in it, and any peculiar odour possessed by it. In infants, examine the contents of the stomach for the presence of milk, food, &c. (see Infanticide, page 360). If there is any reason to suspect poisoning, preserve the contents of the stomach for analysis (see page 124). Note carefully the condition of the mucous membrane (see page 121), and examine for the presence of adhering particles of poison. Any suspicious-looking particles should be picked off with a pair of forceps and separately preserved for analysis.

6. **The Intestines.**—These, after removal, should be laid open along their whole length, preserving, in cases of suspected poisoning, their contents for analysis, with precautions similar to those observed in preserving the contents of the stomach. In the case of infants note if meconium be present, and its position. Note the condition of the mucous membrane, presence of any erosions, ulcers, or perforations, and the condition of the agminate and solitary glands.

N.B.—After completing the dissection of the abdomen, proceed to the dissection of the thorax, should this cavity not have been already dissected (see II. 2).

IV.—The Spine and Spinal Cord.

These should be examined in all cases where it is likely that any signs of injury thereto, or morbid appearance thereof, is to be found. Procedure: divide the integuments down to the bone by an incision in the middle line along the whole length of the spinal column; reflect the integuments on either side and cut away the muscles from the arches of the vertebræ. In carrying out this dissection, note any extravasations of blood in the tissues over the spine, and any fracture of the bones which may be exposed. Then saw through the vertebral arches on either side and remove the detached portions of bone. Examine the outer surface of the exposed dura mater, then slit it open carefully along its whole length, and examine the exposed portion of the pia mater *in situ*. Next pass the finger gently down the cord, noting its consistence; then remove the cord from the body and

complete its examination, making for this purpose transverse incisions through it in several places. Finally, remove the dura mater from the interior of the spinal canal, and complete the examination of the vertebræ for fractures.

V.—The Knee Joint.

In new-born infants this joint should be opened by a transverse incision in front, the lower end of the femur pushed out through the wound, and the cartilage at the end of the bone sliced transversely in fine slices until a pink spot appears in the cut surface; very fine slices are then to be made and the greatest diameter of the bony nucleus ascertained (see pages 348 and 375).

AVERAGE WEIGHTS of the PRINCIPAL ADULT VISCERA.

(According to Tidy.)

—	Male.	Female.
	Oz.	Oz.
Brain	49½	44
Lungs (together)	45	32
Heart (usually about in inches 5 × 3½ × 2½)	9½	8¾
Stomach	4½	A little less than 4½
Liver	50–60	45–55
Spleen	5–7	5–7
Pancreas	2¼–3½	2¼–3½
Kidneys (together)	9	8¾

N.B.—Among natives of India the average weights are probably lower than as stated above.

XXX.—Botanical Characters of Indian Poisonous Plants.

Compiled by Surgeon K. R. KIRTIKAR, *Bombay Medical Service.*

Anacardiaceæ.

Semecarpus Anacardium.—A tree with simple, alternate, coriaceous leaves, entire, large, oblong or oblong-ovate, rounded at apex. Panicles terminal, large, stout. Bracts small, many, caducous. Flowers diœcious, small, numerous, fascicled. Calyx 5-parted; sepals deciduous. Corolla 5-petalled; petals much longer than the calyx, oblong, imbricate. Filaments subulate, inserted at the base of the disc. Stamens in hermaphrodite flowers imperfect. Receptacle erect, fleshy, pear-shaped, smooth. Fruit (marking nuts), a drupe, described in the text.

Anacardium Occidentale.—A small tree with a short crooked trunk; obovate, alternate, short, smooth, entire leaves with a rounded

apex. Panicles terminal, pubescent, bearing both staminate and hermaphrodite flowers, numerously gathered at the tips of several naked, long, alternate pedicels; bracteate. Flowers small, yellowish, with crimson stripes. Calyx 5-partite, nearly to the base, imbricate, erect. Corolla 5-petalled, linear-lanceolate, recurved. Disc filling the base of the calyx, erect. *Male flowers:*—Stamens, 9 usually, all fertile, one more than double the length of the rest, particularly in the sterile flowers. *Female flowers:*—Style long. Fruit one inch long, reniform, on a fleshy peduncle. Both fruit (cashew-nuts) and peduncle (cashew) are edible.

Rhus Acuminata.—A tree 30 ft. high, with alternate impari-pinnate leaves, crowded at apex, elliptic, obovate, oblong, often abruptly cordate, acuminate at length, drooping, glabrous, coriaceous on both surfaces. Panicles axillary, with small numerous flowers. Calyx 5-partite. Corolla 5-petalled, reflected; filaments inserted alternately in the petals. Disc 5-lobed. Fruit, a drupe, one-seeded, in dense drooping panicles on short pedicels.

Asclepiadaceæ.

Calotropis Gigantea.—A smooth erect or hoary shrub, milky, with opposite, broad, sub-sessile or amplexicaul leaves; broad, oblong, or obovate, downy underneath, smooth above; often decussate; entire. The umbels or umbellate cymes on round woolly peduncles, which arise from between petioles, erect. Calyx 5-partite, inferior, glandular within. Corolla 5-petalled, campanulate, naked, valvate, revolute. Stamens gynandrous; filaments connate, cohering round the pistil, capped with a corona consisting of five fleshy radiating scales. Pollen masses waxy. Stigma depressed, 5-angled. Pistil of 2 carpels. Fruit, follicles, short, thick, recurved, often in pair, 3 to 4 in.; seeds ¼ inch long, comose, numerous, lying loose in the follicle.

Calotropis Procera.—Much the same as C. Gigantea. Leaves more oblong and acute. Flowers on terminal or axillary corymbose cymes.

Cryptostegia Grandiflora.—A climbing shrub with opposite short-petioled, polished, entire, obtuse-pointed leaves. Racemes dichotomous with a flower in the fork. Bracts caducous. Flowers, showy, bell-shaped, crimson outside, pink inside. Calyx of 5 lanceolate sepals, with ample thin curled margins. Corolla of 5 petals; campanulate. In the tube of corolla, 5 linear, bipartite scales. Stamens included; filaments short, inserted at base of tube; anthers forming a dome over stigma. Stigma single, large, globose. Ovaries 2, one-celled, each containing numerous ovules. Fruit, a three-sided set of two follicles. Seeds comose.

Tylophora Fasciculata.—A twining erect herb rising in several stems from the root, with approximated, coriaceous, ovate or lanceolate, glabrous, petioled leaves, 1 to 2 in. by ¾ to 1½ in. Umbellate cymes, simple or compound. Calyx and corolla 5-partite. Coronal processes globose. Fruit, follicles.

Convolvulaceæ.

Ipomœa Cærulea.—A tall herbaceous twining hairy annual, with downy, broad cordate, three-lobed leaves, alternate petioled. Flowers on axillary long peduncles; showy, blue or purple, somewhat orange below. Calyx 5-partite, inferior, enlarged in fruit. Corolla gamopetalous, 5-petalled, plaited, hypogynous, funnel-shaped. Stamens epipetalous, anthers included, never twisted. Stigma, globose, bi-lobed. Pistil of 3 carpels, syncarpous, superior. Ovary 3-celled, 6-ovuled. Fruit capsule. Cotyledons plaited.

Ipomœa Turpethum.—A twining plant with a quadrangular winged stem. Leaves variously shaped from broad cordate to arrow-shaped angular. Calyx 5-partite, inferior globular, increasing in size as the fruit matures. Corolla of 5 petals, tubular-campanulate. Stamens epipetalous, anthers soon twisted. Stigma of two round lobes. Fruit a capsule, globose, involved, in a dry translucent calyx, absolutely 4-sided, 4-celled. Cotyledons leafy.

Cuscuta Reflexa.—Parasitic twining herbs, originally germinating from seeds, but soon leaving the ground and transferring themselves by means of suckers to adjacent trees, which serve them as hosts all their future life. Stems succulent, filiform, leafless. Flowers campanulate, in clustered long racemes, minute. Filaments five, very short; anthers oval, appearing above the mouth of the tube. Stigmas 2, large, fleshy. Fruit 2-celled, 4-seeded capsule.

Cucurbitaceæ.

General Characters.—Herbs or shrubs, generally succulent, but not fleshy, climbing by means of solitary, lateral, spiral, simple or divided tendrils, or prostrate, containing watery juice. Annuals growing in the rains, seldom perennial. Leaves petioled, alternate, more or less scabrous, simple, cordate, palmate or lobed, with variously serrated, dentate, or spiked margins. Flowers unisexual monœcious or diœcious; solitary in axils when fertile or barren; when clustered barren, and in racemes or panicles. Calyx monosepalous, 5-toothed, wholly adherent to the ovary (except in Actinostemma). Limb campanulate or tubular, sometimes obsolete; superior in the female flower. Lobes 5, rarely 3, imbricate. Corolla of 5 petals, inserted on the calyx-limb; more commonly monopetalous, sometimes free or nearly so. Yellow, orange, or white. Petal-margins sometimes fringed. Æstivation valvate or involute. *Male flowers*:—Stamens 3, sometimes 2 or 5, epipetalous, distinct, short, thick, monadelphous or triadelphous—2 bundles each containing 2 stamens, and the third but 1 stamen. Connective sometimes prolonged beyond anthers, which are extrorse, 2-celled, usually long and sinuous. *Female flowers*:—Ovary inferior, 1-celled, though usually composed of 3 to 5 coherent carpels; rarely of 1 carpel; spuriously 3-celled from the 3 parietal placentas reflecting towards the circumference. Style terminal, trifid, short; stigmas thick lamellate, lobed or fringed, papillose. Fruit, a pepo: rarely dry, dehiscing variously. Seeds numerous, usually with a leathery horny crustaceous testa, often

thick bordered; embryo straight, filling the seed, exalbuminous cotyledons flat, leafy.

Citrullus Colocynthis.—Tendrils 2 to 3-fid. Flowers all solitary. Stamens inserted below the mouth of the calyx-tube; anthers more or less cohering, usually exserted from the tube; calyx-tube of the male flower short; petals entire, 5, divided quite to the base. Anther-cells conduplicate or sigmoid. Female flowers, usually solitary, never panicled. Ovary villous. Fruit globose, rarely 3 inches in diameter, pulpy inside, woody when dry.

Lagenaria Vulgaris.—Leaves 5-lobed, not divided into distinct leaflets. Petals entire, 5, divided nearly to the base. Calyx-tube of the male flower elongate. Anthers usually included in the tube or nearly so; anther-cells sigmoid. Fruit very large, bottle-shaped, from solitary female flowers. Petioles with two glands at apex; tendrils divided. Ovules numerous, parallel to and near the margin.

Citrullus Amarus.—Var. of C. vulgaris. A trailing herb; does not much differ from the cultivated forms of C. Colocynthis. Leaves deeply 3 to 5-lobed; divisions again lobed or sinuate-pinnatifid, pale or bluish. Pulp mainly formed of enlarged or succulent placentæ. Fruit often 10 inches in diam.

Cucumis Sativus.—Highly scabrous herb; stem twisted; tendrils simple; leaves not deeply divided into distinct leaflets. Stamens 3, inserted below the mouth of the calyx-tube; anthers more or less cohering, usually exserted from the tube; anther-cells sigmoid. Calyx-tube short. Corolla entire, of 5 petals divided quite to the base. Female flowers usually solitary; male flowers solitary or clustered. Fruit spherical or elongate, smooth or tuberculated. Seeds spindle-shaped.

Cucumis Trigonus.—Root perennial. This distinguishes it from C. Melo. Stem scabrid, tendrils short. Leaves deeply palmately-lobed. Fruit of the size of a small hen's egg. (Described in the text.)

Trichosanthes Dioica. — 2–5-fid tendrils. Leaves sinuate-dentate, not lobed. Male peduncles paired. Petals 5, with fimbriate margins. Stamens three, anthers free, 1 one-celled, 2 two-celled, conduplicate. Fruit oblong or nearly spherical, acute, orange-red.

Momordica Cymbalaria.—Root perennial, tuberous, ovoid or turnip-shaped, often irregularly so. Stem slender, smooth, five-sided. Tendrils simple. Male flowers partly racemed. Stamens two, inserted below the mouth of the calyx-tube; anthers more or less cohering—horse-shoe shaped. Female flowers usually solitary.

Momordica Charantia.—Root semi-tuberous, stem hairy. Leaves pubescent on the under-surface. Petiole deeply grooved. Fruit rostate. Seeds when ripe coated with a scarlet mucilaginous aril.

Luffa acutangula, var. Amara.—Leaves not divided into distinct leaflets. Female flowers solitary, axillary, peduncled. Male flowers on long erect axillary racemes; the pedicels have a glandular bract near the base, and are articulated a little above it. Stamens inserted near the mouth of the calyx-tube; anthers exserted free. Stigma 3-lobed. Fruit opening by a stopple.

Luffa Echinata.—Stem 5-sided, slightly scabrous, pilose; tendrils bifid. Flowers small, white, ebracteate; female flowers on very

short peduncles. Style 3-cleft; each cleft 2-lobed. Stopple or operculum without spines. (Fruit described in the text.)

Corallocarpus Epigœa.—A herbaceous plant with a round smooth stem. Tendrils simple. Flowers not large, yellow. Male racemes or pedicels not stout. Anther cells straight. Fruit $\frac{3}{8}$ in., conical, smooth, red berries, circumsciss near the base, containing about six seeds.

Euphorbiaceæ.

General Characters.—Herbs, shrubs, or trees yielding a milky, often acrid, juice. Roots sometimes tuberous. Leaves usually simple, alternate or opposite, rarely divided, palmate or digitate, or compound, with or without stipules. Flowers irregular, often minute, monœcious or diœcious; axillary or terminal; rarely pedunculed; achlamydeous, or with a lobed inferior calyx, with glands inside, or with 3 to 5 distinct or united minute petals. Sometimes the flowers are enclosed in a calyx-like involucre. *Male flowers:*—Stamens one or more, distinct or united; rarely a single stamen on a pedicel with which the filament articulates. Anthers 2-celled, often two distinct lobes, with or without a connective at the top end of filament. Filaments sometimes polyadelphous, geniculate. *Female flowers:*—Ovary superior, 3-carpelled; rarely 2 or more carpels more or less united. Style usually present, one for each carpel, entire or bifurcating at the position of stigma; sometimes each bifurcation bifurcating again. Ovules pendulous from inner carpel-angles. Funicle often thickened. Fruit, a capsule or drupe. Seeds 1 to 2 in each carpel; often with an axil; some carunculate. Embryo in a fleshy albumen, very rarely exalbuminous.

The Tribe of the Euphorbias.

General Characters.—Some have stems resembling the succulent Cactaceæ, from which they differ in possessing milky juice. Flowers involucrate, monœcious; involucres calyciform, enclosing male and female flowers. Flower-heads resemble single flowers, consisting of an involucre, within which there are several single stamens constituting the male flowers, and one central female flower in the shape of a single pedicellate pistil. Ovary-cells with one ovule.

Euphorbia Neriifolia.—Stem armed with pairs of stipular spines; branches round, with five more or less spirally twisted ribs. Leaves apical, in clusters or whorls.

Euphorbia Antiquorum.—Stem armed with pairs of stipular spines. Branches with five sharp prominent angles. Peduncles solitary or in pairs a little above the spines; usually three-flowered, the central flower-head fertile. Leaves minute or wanting.

Euphorbia Cattimandoo.—Stem armed with stipular spines in pairs; branches with five sharp prominent angles, which are deeply sinuate. Leaves cuneate, mucronate. Flowers after its leaves have fallen.

Euphorbia Tirucalli.—Stem unarmed, cylindrical. Leaves linear-lanceolate. Branches green, cylindrical, succulent, numerous;

capped with alternate remote leaves. Flower-heads small, numerous; terminal or axillary.

Euphorbia Hirta.
E. Thymifolia.
E. Parviflora.
} Generally red-tinged, recurved, unarmed, prostrate herbs. More or less pubescent. N. L. Burmann distinguishes E. Parviflora from the others by describing it as sub-dichotomous, and the other two as dichotomous. Calyx and corolla of four semi-lateral parts each. Roots contorted and thickened more or less. E. Parviflora has small rounded leaves, the other two have larger oblong-ovate acuminate leaves.

Euphorbia Rothiana. — Unarmed, erect, herbaceous plant. Leaves oblong-lanceolate entire. Flowers bracteate in umbels. Involucre ciliated at margin, linear-oblong. Involucels ovate-cordate. Inside of calyx woolly. Stamens furnished at base with a filiform pubescent scale.

Croton Tiglium.—A small tree with a crooked glabrous ash-coloured trunk; leaves ovate-cordate, 3 to 5 inches by 2 to 3 inches. Greenish-yellow terminal racemes, on the upper part of which are numerous male flowers. Stamens 15 to 20, woolly, unconnected. Female flowers single, at the lower part of the raceme, apetalous, longer than the male flower; some small glands round the ovate base. Fruit, a 3-cornered capsule, ¾ inch long.

Jatropha Curcas.—A hedge plant flowering throughout the year. Trunk irregular. Epidermis peeling off in scales. Leaves smooth, broad cordate, five-angled, smooth, petioled, exstipulate. Panicles terminal or from exterior axils. Male flowers at the extremities of the ramifications on short articulated pedicels; female flowers in their divisions, not articulated; bracts small. Calyx 5-lobed. Corolla 5-petalled, campanulate, hairy. Five glandular bodies round base of filaments. Filaments 6, the central very thick, columnar, surrounded by the remaining 5 filiform ones; anthers sagittate; styles 3, short; stigma bifid, hairy. Fruit, a capsule, 3-celled, 3-seeded.

Jatropha Multifida.—A succulent ornamental garden plant; leaves digitately multifid, with small red flowers in umbellate cymes, borne on long erect succulent and showy peduncles. Calyx of 5 sepals, gamosepalous, inferior. Corolla 5-petalled, campanulate. Stamens 10, monadelphous, hypogynous. Pistils of 3 carpels, syncarpous.

Jatropha Glandulifera.—A stout shrub dichotomously branched. Leaves of a peculiar green and deep crimson, palmate, alternate, petioled; lobes 3 to 5, oblong; ribs beautifully crimson. The margins of leaves, the ventral borders of petioles, the stipules, the bracts, all capped with tufts of bristles ending in a small sticky gland. Panicles few-flowered, terminal. Male flowers scarlet-crimson. Calyx 5-cleft to the base, bristled. Corolla campanulate, conjoined towards base; woolly inside there. Filaments eight, conjoined, having green glands at base; anthers twin. Female flowers have a calyx and corolla as in the male. Styles three, with enlarged ragged stigmas. Fruit, 3-coccous capsule.

Lebidieropsis Orbicularis.—A large shrub or small tree with scanty light green foliage. Bark blackish, very rough; wood reddish, durable but brittle. Leaves 2 to 4 ins., reticulate-veined. Branchlets,

petioles, calyx, and bracteoles hairy. Flowers yellowish-green in clusters, monœcious or diœcious, axillary, subsessile; petals minute. Stamens five, inserted on a central column on a broad circular disc. Styles 3-fid. Fruit, a capsule, 1 inch long.

Liliaceæ.

Urginea Indica.—(Bulb described in the text.) Leaves narrow, radical, tapering from base, ensiform, flat, glabrous, 2 to 18 in. long. When in bloom, destitute of leaves. Scape erect, round, naked. Flowers in erect racemes, remote, long-pedicelled, drooping. Sepals linear, equal; bracts minute, caducous; filaments filiform. Capsules elliptic, many-seeded. Seed compressed, orbicular, broadly winged, bright shining black.

Gloriosa Superba.—(Root described in the text.) An annual herbaceous climber flowering in the rains. Leaves lanceolate; midrib ending in a coiled recurved stiff tendril. Flowers showy, scarlet, green and yellow mixed. Calyx none. Corolla six-petalled, revolute. Petals lanceolate, with wavy margins, 2 to 3 inches long, scarlet at base, yellow at top. Style oblique. Ovary three-celled, superior. Fruit, a capsule, three-valved, many-seeded.

Thymelaceæ.

Daphne Papyracea.—A tall Himalayan shrub, bi- and trifurcate. Youngest branchlets slightly pubescent. Leaves alternate, entire, smooth, sub-coriaceous; 3 to 5 inches long, narrowed into a short marginate petiole. Flowers 6 to 12 in terminal heads, scented, sessile, numerously bracteate. Perianth of four leaves, gamophyllous, inferior, tubular, pubescent. Stamens 8, half of which epiphyllous. Fruit, ovoid, succulent, red or orange when ripe. Seed solitary, pendulous, exalbuminous.

Lasiosiphon Speciosus.—A shrub with scattered very shortly petioled willow-looking leaves, lanceolate, oblong, rather acute; heads of flowers terminal, surrounded by an involucre of oblong rather hoary leaflets; calyx hairy; the lobes ovate-obtuse with five linear bifid scales half their length.

Aroideæ.

Arum Montanum.—A stemless plant. Root a cylindric tuber. Leaves cordate, repand, polished, simple. Spadix nearly as long as the hood-like, coloured spathe, with female florets round the base; male about the middle and naked above. Anthers many-celled.

Arum Lyratum.—A stemless plant. Root a tuber of turnip or potato shape, pretty smooth. Leaves radical, one, two, or three, petioled, compound, tripinnatifid; divisions very unequal, the exterior one larger than the inferior ones. Leaflets lyrate. Petioles one or two feet long, swelled and sheathing at the base. Berries of the size of a small jambul, red, smooth, and one-seeded.

Arum Sylvaticum.—Root perennial, tuberous, nearly smooth.

Stem none. Leaves radical, super-decompound, thrice two- or more lobed; one or two petioled; lobes pinnatifid; segments smooth, lanceolate. Leaf 18 to 30 inches each way. Petioles smooth, winged, 1 to 2 feet; scape erect, round; spathe one-third the length of the spadix, opening at or near the apex only. Stamens numerous, occupying about one-fourth of the lower portion of the club, just within the apex of the spathe, singly sessile and roundish with two polliniferous cells which open on the apex. Ovaries numerous, surrounding an inch of the base of the spadix. Style short; stigma three-lobed, glutinous, yellow. Berries one-, two-, or three-seeded.

Papaveraceæ.

Argemone Mexicana.—An erect prickly herb almost perennial, greenish-glaucous; juice yellow, thick. Leaves amplexicaul; midrib spiked on the under surface; margin sinuous, spiked. Flowers bright yellow, solitary, terminal. Sepals 2 to 3, inferior, caducous, spiked at apex, hood-like. Petals 4 to 6, hypogynous, smooth. Stamens indefinite, hypogynous. Ovary one-celled. Stigma, crimson, quadrangular, channelled diagonally. Style short, if any. (Fruit described in the text.)

Menispermaceæ.

Cocculus Indicus.—Perennial, twining, scandent shrub. Leaves ovate or ovate-cordate, 4 to 8 inches long, acute, glabrous above, hairy beneath on the nerve-axils. Panicles on old branches 1 to 1½ foot, pendulous. Flowers ¼ in. diameter, glabrous. Sepals deciduous, 6 with two appressed bracts; no petals. *Male Flowers:*—Anthers sessile on a stout column, 2-celled, bursting transversely. *Female Flowers:*—Staminodes 9, clavate, 1-seriate. Ovaries three on a stout gynophore; stigma subcapitate, reflexed. Drupes ⅜ inch, black, glabrous. (Seeds described in the text.)

Plumbagineæ.

Plumbago Zeylanica.—Perennial garden shrub, many-jointed, declining, ramous. Leaves alternate, short-petioled, simple, ovate, waved, entire, 2 to 3 inches long; amplexicaul, channelled or subwinged, exstipulate. Racemes axillary, terminal, covered with short viscid hairs. Bracts with sticky hairs, three-fold. Calyx 5-toothed, gamosepalous, gibbous, inferior. Corolla 5-petalled, gamopetalous, hypogynous, funnel-shaped. Stamens 5, hypogynous. Stigma 5-cleft. Ovary syncarpous, of 5 carpels, superior.

Plumbago Rosea.—Perennial shrub. Leaves ovate-oblong, smooth. Resembles in general characters P. Zeylanica. Chief differences: racemes longer, flowers smaller, more distant from each other, and of thicker texture. Bracts three-fold, one-flowered, equal, smooth. The inner or upper two bracts united between the flower and rachis. Base of style hairy. In P. Zeylanica the outer bract is ten times larger than the lateral one.

Rubiaceæ.

Randia Dumetorum.—A small thorny tree. Leaves obovate, cuneate at base, 1 to 2 inches, narrowed into a short petiole. Flowers 2 to 3 on a peduncle, sometimes solitary. Calyx with short stiff hairs; teeth variable, sometimes spathulate; 5-sepalled, superior, gamosepalous. Corolla, 5-petalled, gamopetalous. Stamens 5, epipetalous. Fruit, a berry, globose or ovoid, $\frac{3}{4}$ to $1\frac{1}{2}$ inch long, glabrous or pubescent, smooth or obscurely ribbed, yellow with a thick pericarp. Seeds compressed, embedded in a pulp.

Rutaceæ.

Ruta Angustifolia.—A strong-smelling herb, 2 to 3 ft., glaucous-green. Leaves alternate, opposite, triangular-ovate, decompound (pinnate, tri- or unifoliate); dotted with translucent glands, containing oil. Corymbs spreading; flowers greenish-yellow, bracteate. Sepals triangular, acute, 4 to 5-lobed. Petals 4 to 5, ciliate, concave. Stamens twice as many as petals. Capsule obtuse, deeply 4 to 5-lobed. Ovules many.

Sapindaceæ.

Sapindus Emarginatus.—A tree with paripinnate leaves, exstipulate, subverticillate, 5 to 12 inches long. Leaflets 4 to 6, elliptic, oblong, acuminate or emarginate, glabrous above, puberulous beneath. Sepals 5. Petals 4 to 5, oblong or ovate, narrower and longer than sepals. Edge of petals hairy. Stamens 8; anthers apiculate, oblong. Disc concave with a raised fleshy hirsute edge. Panicles pubescent, rust-coloured. (Fruit described in the text.)

Combretaceæ.

Terminalia Bellerica.—A tree with rusty pubescence on young branchlets and calyx. Leaves approximate when young, broad, elliptic, 3 to 8 inches long; base often unequal, apex obtuse. Flowers on 3 to 6 inches long spikes; sessile, offensive; male and bi-sexual mixed; bracts linear, caducous. Free part of calyx cup-shaped, cleft half-way into five acute triangular segments, woolly inside with long brown hairs. Filaments inserted below the segments and twice their length. (Fruit described in the text.)

Ficoideæ.

Trianthema Monogyna.—Stem prostrate, glabrous; leaves opposite, red-margined, obcordate and oblong alternately. Flowers solitary; calyx-tube scarious, thin, closely sheathed by the base of the petiole. Stamens 15 to 20, inserted near top of calyx-tube. Corolla 5-petalled—(*N.B.*—Hooker says petals are absent),—daggered under the top. Style single. Capsule inferior, circumcised, $\frac{1}{5}$ inch, scarious below; beak exserted, coriaceous, somewhat mitriform, adnate to the enclosed seed; seeds in lower part 3 to 5, black, reniform, rough.

Trianthema Pentandra.—Stem papillose or nearly glabrous. Leaves oblong or elliptic. Flowers in sessile clusters. Calyx lobes ovate, often scarious on the margin; bracts scarious. Styles two. Capsule $\frac{1}{8}$ inch; beak consisting of two lanceolar portions, acute upwards, coriaceous, closely adnate to the included seed. Seeds in the lower half of the capsule 2, dull black, roughly puberulous.

Leguminosæ.

Abrus Precatorius.—SUB-ORDER PAPILIONACEÆ.—A climbing shrub. Root hard, woody, sweet, and mucilaginous. Branches many, slender. Leaves 2 to 3 inches long, paripinnate, stipulate. Leaflets opposite, sub-sessile, 8 to 15 pairs, smooth, entire, small, deciduous, linear oval, obtuse. Racemes long-peduncled, axillary, solitary. Flowers many, crowded. Calyx $\frac{1}{12}$ inch, thin, silky, 4-lobed, campanulate. Corolla 3 to 4 times as large as the calyx, red or white. Stamens 9, united in a slit above; the tenth absent. Anthers ovate. Stigma blunt. Fruit a legume. (Seed described in the text.)

Urticaceæ.

Cannabis Sativa.—A diœcious herb. *Male flowers:*—In glomerules; 5-parted calyx; without corolla. *Female flowers:*—In spikes; two styles, two stigmas, pubescent, elongate filiform. Female calyx consists of a single-folded sepal, entire, opening sideways. Fruit caryopsis. Seed solitary, pendulous.

Solanaceæ.

Datura Alba and Datura Fastuosa.—Perennial herbs, with white and purple flowers respectively. Leaves unequally ovate; margins repand-lobate, pubescent. Pedicels solitary. Flowers axillary. Calyx of 5 sepals, gamosepalous, long tubular, inferior. Corolla of 5 petals, gamopetalous, hypogynous, infundibuliform, plaited, wide-mouthed. Stamens 5, epipetalous, attached near the base of the tube. Filaments filiform; anthers included, linear, longitudinally dehiscent. Ovary 2-carpelled, syncarpous, superior. Style filiform; stigma 2-lobed. Fruit a capsule, spuriously 4-celled, prickly, on a thick torus, irregularly dehiscent near the apex. (Seeds described in the text.)

Loganiaceæ.

Strychnos Nux-Vomica.—A middle-sized tree, often with a thick crooked trunk. Leaves 3 to 5 inches long, coriaceous, opposite, broadly ovate, entire; stipules interpetiolar. Corymbose cymes, small, rounded, terminal. Flowers small, pubescent, greenish-white. Calyx of 5 sepals, gamosepalous, inferior, permanent. Corolla of 5 petals, hypogynous, infundibuliform. Stamens 5, epipetalous. Anther-cells half in, half out of the tube. Ovary syncarpous, 2-celled, superior. Stigma small, capitate. Fruit, an orange-sized amphisarca of orange colour, with a hard shell containing discoid seeds embedded in a soft pulp.

Strychnos Colubrina.—A scandent shrub with tendrils often in pairs. Leaves 4 by $1\frac{3}{4}$ inches; base cuneate or obtuse. Cymes 1 to 2 inches; calyx puberulous; corolla $\frac{1}{10}$ inch; tube hardly longer than calyx; lobes villous within. Anther-cells oblong, glabrous, or nearly so. Ovary glabrous below, fulvus hairy above. Style as long as the ovary. Fruit, an amphisarca $\frac{2}{3}$ inch diameter, crustaceous. Seeds 1 to 2, discoid.

Ranunculaceæ.

Aconitum Ferox.—A perennial herb; stem erect, simple below. (Root described and figured in the text.) Flowers large, pale, dirty blue, in racemes; bracteate. Sepals petaloid, 5, posterior (helmet) vaulted, twice as long as high. Stamens many. Fruit consists of 5 follicles, erect, usually densely villous, sometimes glabrous.

Apocynaceæ.

Plumieria Acutifolia.—A large shrub or tree, milky. Branches blunt, truncated, several, with tufts of aerial rootlets. Leaves crowded at the club end of the bud in irregular whorls, sprouting after the flowers appear at the apices; acuminate, lanceolate; 1 foot long, 3 inches broad, entire; petioles channelled. Flowers in verticillated corymbiform racemes. Æstivation twisted; bracts broad, deciduous. Calyx small, 5-fid, gamosepalous, inferior, fleshy, eglandular. Corolla 5-petalled, hypogynous, salver-shaped; throat naked. Carpels 2, superior. Stamens 5, small, epipetalous, at the bottom of the tube; anthers sagittate. Style short; stigma two-lobed. Fruit rarely seen in Western India.

Nerium Odorum.—A shrub or tree; juice acrid, watery. Stem erect, quadrangular or triangular at growing apices. Leaves entire, opposite, or whorled at apex, 4 to 6 inches, linear-lanceolate, smooth, thick, coriaceous, acuminate. Flowers $1\frac{1}{2}$ inch diameter, sweet, rose-coloured, yellowish white or deep crimson; terminal, solitary or in umbellate cymes. Calyx of 5 sepals, erect, lanceolate. Corolla funnel-shaped, of 5 petals. Mouth of tube variously crowned. Stamens 5; filaments very short, thick, inserted on the mouth of corolla-tube. Anthers sagittate, united into a dome over the hour-glass-shaped stigma. Connective prolonged into a feathery process, wavy, twice as long as the anther. Fruit 2-follicled, each follicle 2 to 6 inches long. Seeds numerous, comose.

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